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THE ALLERGIC ASPECT OF VASOMOTOR RHINITIS

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The allergist and the rhinologist often are confronted with cases of nonseasonal coryza uncomplicated by infection of the nasal passages or paranasal sinuses. The characteristic pathologic picture is that of a pinkish gray boggy mucosa with rhinorrhea and serous discharge, the bogginess resulting from epithelial hyperplasia. The patient complains of stuffiness of the nose with periods of complete obstruction. He usually gives a history of violent sneezing, worse at certain times, as on getting up in the morning or going to bed, and of being awakened at night by inability to breathe through his nose. This is the type of coryza that is generally labeled vasomotor rhinitis.

In the early stages the patient is apt to describe his condition as a "head cold," thus suggesting the possibility of a basic infectious state of the nasal passages and sinuses, or perhaps a superimposed infection. It is not uncommon for nasal allergy and infection to coexist, since the obstruction and impairment of aeration caused by allergic edema favor the development of infection in the nasal mucosa. The patient may also speak of his condition as "sinusitis," which he believes to flare up from time to time.

The differential diagnosis can easily be made by examining the nasal secretions for bacteria and eosinophils and the blood for eosinophilia and by transillumination and roentgen examination of the accessory sinuses. At the same time, mechanical obstructions in the form of deflected septums, spurs or polypi and evidence of cystic changes in the nasal and paranasal structures should be looked for. If no signs of infection or other local pathologic changes can be discovered, but the eosinophil cells in the blood and the nasal secretions are found to be increased and there is also a family history of allergy, the case may be regarded as one of vasomotor rhinitis of probable allergic origin.

As is well known, this malady often shows stubborn resistance to therapy and requires extensive and exhaustive study from the allergic

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point of view. In determining the diagnosis the following possible causes should be investigated. 1. All specific allergenic agents known to give rise to this condition. (These will form the basis of the present discussion.) 2. All nonspecific factors. These usually can be elicited from the history alone. 3. All endocrine and physiologic dysfunctions that may play a primary or a secondary part in causing the disease.

To rule out the possibility of nonallergic etiologic factors coexisting with the allergic ones, the allergist should have the patient examined for foci of infection or any intrinsic conditions relating to the nasopharynx that may have escaped his own observation. Woodward and Swineford [1] have advocated close cooperation between the allergist and the rhinologist for optimum results in both the diagnosis and the treatment of allergic rhinitis.

It is my purpose to discuss only the uncomplicated type of vasomotor rhinitis and the specific allergenic agents that may be responsible for its clinical manifestations. The nonspecific factors consist largely of substances that cannot be used for testing but which have been found to exert some etiologic influence on the condition. In this group are vapors, smoke and gases; fumes of varnish, lacquer, tar and dyes; odors of food and perfume; chemical irritants, such as acids, alkalis and oils; physical agents, including heat, cold, light, steam, dampness, wind, rain, snow, chilling or overheating of the body and perspiration.

The specific allergens fall into two major groups, inhalants and foods, both of which occasionally are involved in a single case of vasomotor rhinitis. Tables 1 and 2 show the chief allergens in both groups, together with the specific excitant content of each in terms of units of protein, nitrogen or of total nitrogen per cubic centimeter. It represents the minimum number of test substances necessary to afford a clue to the possible causes of sensitivity. The substances listed are used in this dilute form for preliminary testing. When the results are negative, more concentrated extracts should be tried. Many other substances may be found necessary for testing any particular patient.

The first step in allergic diagnosis is an attempt to ascertain the causative agent by means of intradermal injections of the inhalant allergens. One begins with a weak dilution of the allergenic extract, and if the reaction is negative, one continues with progressively stronger concentrations. It is in this technic that the experienced allergist has the best hope of discovering the responsible agent, since it enables him to note degrees of sensitivity that ordinarily would be missed. The sum total of moderate sensitivities, which singly might be disregarded, may well be the cause of symptoms. If doubtful results are obtained by

1. Woodward, F. D., and Swineford, O.: Allergic Rhinitis: Analysis of One Hundred and Twenty-Eight Cases in Which the Rhinologist and the Allergist Collaborated, Arch. Otolaryng. **34**:1123 (Dec.) 1941.

this method of direct cutaneous testing, confirmation should be sought by the passive transfer test.

Next, one tries the food allergens, but it must be remembered that in adults the cutaneous reaction is not a reliable index of clinical sensitivity. It must be supplemented by a thorough study of the food diary or by the method of eliminating suspected foods. Young and middle-aged persons are most frequently affected by vasomotor rhinitis, and it is in these groups that the cutaneous test for food allergy most often fails to yield the desired information. For example, a positive reaction may

TABLE 1.—*Inhalant Allergens Used for Diagnostic Test Studies*

Substance	Units of Protein Nitrogen per Cc.*	Substance	Units of Protein Nitrogen per Cc.
Dust	Full Strength	Dog epithelium	100
Tobacco	100	Cat epithelium	100
Feathers	100	Rabbit epithelium	100
Horse serum	1-100	Horse epithelium	100
Pyrethrum	100	Raw silk	100
Le Page's glue	10	Orris	100
Cottonseed	100	Wool	100
Kapok	100	Flaxseed	100
Fungi (yeasts, molds, smuts)			

* One unit = 0.00001 mg. of protein nitrogen.

TABLE 2.—*Food Allergens Used for Diagnostic Test Studies*

Substance	Mg. of Total Nitrogen per Cc.	Substance	Mg. of Total Nitrogen per Cc.
Egg white	0.0001	Lima bean	0.1
Milk (casein, whey)	0.1	Orange	0.1
Wheat	0.1	Green pepper	0.1
Beef	0.1	White potato	0.01
Chicken	0.1	Rice	0.1
Pork	0.1	Green pea	0.1
Fish	0.01	Banana	0.1
Shellfish	0.01	Chocolate	0.05
Corn meal	0.01	Peanut	0.001

mean only that the patient has retained cutaneous sensitivity to some food to which he formerly was allergic but can now ingest without clinical symptoms. Or, a positive cutaneous reaction may indicate only a potential clinical intolerance instead of one actually present. On the other hand, the skin tested may prove negative to a food which the patient knows causes symptoms within six to twenty-four hours after ingestion. Such cases may be explained by the fact that the patient is allergic only to the split products of the food, which become active in the late stages of digestion, while the cutaneous test naturally is made with the whole, unaltered food, to which he is immune.

Despite these limitations, however, the cutaneous test for food allergy should be made and the result recorded for use as a clue; before any food can be incriminated, however, the test must be supported by the food diary or the results of elimination diets.

THREE IMPORTANT GUIDES TO SUCCESSFUL MANAGEMENT

Complete History.—This should include the patient's own description of his home and occupational environment, of his home, business and social contacts, of cosmetics used, of clothing worn and of hobbies, sports and other activities. Data on all these items are necessary in order that one may arrive at an understanding of the patient's condition. Unless the history is all inclusive, the causative factors may be difficult to discover or may be overlooked entirely.

Clinical Observation.—The history given by the patient should be confirmed and supplemented with an exhaustive study of home and occupational conditions by a social service nurse (or home investigator in the case of private patients). The work of this assistant, who should have been trained for the survey of homes with respect to allergens, is extremely important for all classes of patients. Fifteen to thirty minutes should be devoted weekly to a check-up with the patient as to his mode of living and all contacts, casual or otherwise. His food intake should be carefully scrutinized by means of the food diary or the elimination diet. His visits to friends' homes, his whereabouts during free time, his travels and pastime contacts and all his various interests and activities should be carefully analyzed for a clue to his symptoms.

Exhaustive Tests.—The cutaneous tests must cover all possible materials to which the patient may be allergic. No potential irritant should be considered unimportant, regardless of how remote the possibility may be. For example, the vasomotor rhinitis of 1 patient was finally traced to iodized salt. The tests should be made not only with all possible irritants but with various concentrations of each, as already outlined. It is only when the patient fails to react to the strongest extract that he can be considered cutaneously insensitive to that particular material.

TREATMENT: DOSAGES AND NUMBER OF ALLERGENS TO USE

Exhaustive and intensive therapy has helped in many cases to clear up vasomotor rhinitis which at first appeared to be intractable. Intensive therapy consists of rapidly working up to a dose (or the dose of a particular allergen considered optimal for a particular patient) by increasing the frequency of treatments to twice or three times a week and then continuing with the maximum dose at short intervals.

It also is necessary to use every kind of allergen to which the patient has shown sensitivity. Even when his contacts with some of these are casual and infrequent, their cumulative effect may be enough to precipitate severe symptoms. It is my rule to treat with all such casual contact allergens when a possible etiologic relation to the symptoms may exist. For instance, if a patient is found to be sensitive to cotton or horse hair, it is not enough to remove cotton or horse hair from the bedding, wearing apparel and other articles of his environment; it is also necessary to immunize him to the substance intensively by the administration of optimal top doses. The same principle applies, of course, to other irritants like tobacco, orris root, epitheliums of other animals and so forth.

Is is only by carefully carrying out the aforementioned procedures that one may hope for satisfactory results from the treatment of allergic vasomotor rhinitis.

CLASSIFICATION OF PATIENTS

Among a large series of patients with vasomotor rhinitis, uncomplicated by any other rhinologic condition who have been studied in the outpatient department of Gouverneur Hospital, the following groups have been distinguished:

Group 1 (patients showing sensitivity to dust, animal epitheliums, orris root, insecticides, tobacco and miscellaneous inhalants).—In this group, dust was found to be by far the most frequent cause, with animal epitheliums next, and the rest in the order given. Treatment with the allergen and the removal of this factor from the patient's environment were effective in clearing up the symptoms.

Group 2 (patients sensitive to occupational irritants).—In this large group the history showed onset of symptoms at varying periods after the patient's employment in a certain occupation. There was either preexisting sensitivity to allergens encountered in the work or gradual sensitization due to continued contact with them. The following types of skilled and unskilled workers, most of whom are included in table 3, are subject to sensitization by the materials of their trade:

(*a*) Furriers. Workers in contact with animal skins may become sensitive to the undyed furs or to dyes and chemicals employed in the preparation of furs. It is well to bear in mind also that rabbit and even cat fur often passes under a more impressive trade name, such as "lapin," "mole," "nutria," and even "fox" or "mink."

A large number of the patients who had worked for some time with different kinds of fur, including genuine raccoon, muskrat, silver fox, Persian lamb and mink (both dyed and undyed), began to have nasal symptoms. Constant contact with the furs or the dyes produced sensitization with slowly developing vasomotor rhinitis, culminating after a year or more in severe nasal blocking and rhinorrhea.

Concentrated extracts of raccoon, muskrat and silver fox gave positive cutaneous reactions, but I believe the dyes containing paraphenylenediamine (ursol D) and paraaminophenol (ursol P) played the most prominent part in the production of symptoms in this group.

Treatment with extracts of animal epitheliums was not encouraging except when supplemented with frequent vacations from work. One week of every eight or ten, or five to eight full weeks' vacation a year, is considered minimal.

(*b*) Upholsterers. The chief excitants encountered by this group are feathers, cotton, kapok and animal hair, but a number of other substances in this trade may give rise to symptoms. The causative factors often are difficult to identify, but when they are found, treatment may enable the patients to continue their work.

(*c*) Clothing Workers. These workers were found to be affected by silk, wool, cotton, linen and shop dust. The results of treatment were poor, and the patients were required to give up their occupation.

(*d*) Hatmakers. Causative allergens in hat manufacture are wool, silk, cotton and rabbit fur. Sternberg and Sorrell [2] found a hat maker to be sensitive to banana oil used in the process of bleaching. The scratch test was positive, but treatment with the oil was contraindicated, so that a change of occupation was the only alternative.

(*e*) Cosmeticians. Orris root is the chief cause of symptoms in these workers, but other materials such as acacia, tragacanth, karaya, linseed and quince seed in permanent wave lotions have been reported as excitants of vasomotor rhinitis.

Dust and orris root were found to be the chief offenders among barbershop workers, and some of these were able to continue in their occupation after receiving treatment.

(*f*) Woodworkers. Imposing lists have been made of the various woods that may cause contact dermatitis, but reports of nasal allergy from this cause are less frequent. However, vasomotor rhinitis has been shown by the nasal test to result from birch wood, and intracutaneous tests with other wood extracts have given positive reactions. According to Bahn,[3] treatment in these cases is not entirely satisfactory.

(*g*) Jewelers. This group was found to be sensitive to solder dust and cuttlefish bone. Treatment was unavailing unless the patient gave up his work.

(*h*) Chemists and druggists. Numerous drugs and chemicals give rise to vasomotor rhinitis among pharmacists and laboratory workers.

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2. Sternberg, L., and Sorrell, A. H.: Occupational Asthma and Vasomotor Rhinitis, New York State J. Med. **41**:1649 (Aug. 15) 1941.

3. Bahn, K.: Beitrag zur Frage der Allergie bei Holzsägereiarbeitern, Klin. Wchnschr. **7**:1963 (Oct. 7) 1928; cited by Sternberg and Sorrell.[2]

Ipecac, lycopodium, podophyllin, rhubarb, castor bean, vanilla, caroid (a preparation of papaw) urease and peptone have been incriminated. The nonprotein drugs and chemicals seldom form reagins, and for this reason cutaneous tests for sensitivity most often prove negative. The following case, reported by Kern,[4] is highly exceptional:

> The patient was a laboratory worker suffering from vasomotor rhinitis. A violent cutaneous reaction resulted from a scratch test with phthalic anhydride in crystalline form, and even the reaction to a dilute alcoholic solution of this substance (1:1,000) was strongly positive. Passive transfer tests also were positive. In this instance the symptoms were caused by inhalation of phthalic anhydride in dust that came into the laboratory where the patient worked, and it was necessary for him to transfer his work to another location.

Since cutaneous tests with drugs and chemicals are rarely positive, the diagnosis depends more on the patient's occupational history and the presence of eosinophilia as indicated by a superabundance of eosinophilic cells in the secretions. Treatment with the causative allergens in these cases must be attempted with great caution for fear of constitutional reactions.

In the patients of this group in the present series the symptoms were caused chiefly by inhaled chemical gases and fumes, and in such cases treatment is of no avail; the patients must be removed from their work.

(*i*) Food handlers. The potential allergens in food storage warehouses, restaurants, grocery stores and other places of business in which foodstuffs may be inhaled are almost unlimited. Wittich[5] reported allergic rhinitis among sorters of peas and beans as a result of the infestation of the stored material by the Mexican bean weevil. Sternberg and Sorrell[2] observed vasomotor rhinitis in a restaurant employee, from the fumes of coffee, and in a warehouse worker, from the dust of cacao beans and chocolate.

Inhalation of various spices is another frequent cause of symptoms. I[6] observed vasomotor rhinitis in a bookkeeper employed in a wholesale spice store, who sometimes handled the products. By intradermal tests with the different spices, I finally discovered that paprika produced a positive reaction. On the patient's removal from contact with paprika the symptoms immediately ceased. Among grocers shop dust was found to be the most frequent irritant. After immunization to dust, some of these patients were able to continue with their work.

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4. Kern, R. A.: Asthma and Allergic Rhinitis Due to Sensitization to Phthalic Anhydride, J. Allergy **10**:164 (Jan.) 1939.

5. Wittich, F. W.: Allergic Rhinitis and Asthma Due to Sensitization to the Mexican Bean Weevil (Zabrotes subfasciatus, Boh.), J. Allergy **12**:42 (Nov.) 1940.

6. Gelfand, H. H.: Vasomotor Rhinitis and Asthma Due to Paprika, J. Allergy **12**:312 (March) 1941.

TABLE 3.—*Analysis of Fifty-Two Cases of Vasomotor Rhinitis*

Patient	Name	Sex	Age	Occupation	Duration of Symptoms	Group *	Allergens		Condition at Time of Report
							Inhalants	Foods	
1	J. W.	M	51	Grocer.....	6 mo.	1 and 2	Dust	.....	Improved
2	B. V.	M	24	Grocer.....	4 yr.	1 and 3	Dust, dog hair	Eggs	Relieved
3	R. B.	M	25	Station.... agent	5 yr.	1 and 2	Dust	.....	Fair
4	M. S.	M	40	Furrier.....	2 yr.	1 and 2	Dust, furs	.....	Fair
5	J. F.	M	54	Furrier.....	2 yr.	1 and 2	Dust, furs	.....	Fair
6	N. E.	M	38	Baker.....	1 yr.	1 and 3	Dust, feathers	Fish	Relieved
7	J. S.	M	37	Grocer.....	7 yr.	1 and 2	Dust, orris	.....	Changed job
8	F. V.	M	21	Food handler	1 yr.	1, 2 and 3	Dust, horse epithelium, ephedrine	Cinnamon	Fair
9	A. T.	F	..	Pencil manufacturer	1 yr.	4	Idiopathic	.....	Referred to rhinologist
10	A. F.	M	..	Physician	1 yr.	1 and 3	Dust	Whey, milk	Relieved
11	J. M.	M	43	Clothing worker	5 yr.	1, 2 and 3	Dust, wool, cotton	Fish	Fair
12	R. L.	M	35	Grocer.....	2 yr.	4	Idiopathic	.....	Referred
13	R. S.	F	60	Housewife.	16 yr.	4	Idiopathic	.....	Referred
14	D. M.	M	46	Feather worker	4	1 and 2	Dust, feathers	.....	Fair
15	V. P.	F	29	Clerk.....	2 yr.	4	Idiopathic	.....	Referred
16	F. P.	F	30	Bakery clerk	1 yr.	1 and 2	Wheat, rye, feathers	.....	Stopped treatment
17	M. S.	M	44	Clothing worker	1 yr.	1, 2 and 3	Dust, wool, cotton	Wheat	Unimproved, stopped treatment
18	P. S.	F	39	Housewife.	10 yr.	1	Dust, animal epithelium	.....	Improved
19	B. D.	F	34	Dry goods worker	1 yr.	1, 2 and 3	Dust, wool, furs	Apples	Improved
20	I. A.	M	47	Ladies' specialties	8 yr.	1 and 3	Dust	Milk products	Improved
21	M. A.	F	36	Clothing worker	14 yr.	1 and 2	Dust, cotton	.....	Improved
22	N. S.	M	21	Baker.....	2 yr.	1 and 2	Dust, rye, wheat	.....	Relieved
23	P. G.	M	31	Clothing worker	3 yr.	1 and 2	Dust, wool	.....	Fair
24	L. F.	F	40	Housewife.	1 yr.	4	Idiopathic	.....	Referred
25	M. B.	M	20	Furrier.....	6 mo.	1 and 2	Dust, furs	.....	Improved
26	H. W.	F	43	Housewife.	7 yr.	1	Dust, perfumes, ephedrine	.....	Improved
27	H. E.	F	18	Secretary..	2 yr.	1	Dust, tobacco	.....	Improved
28	J. K.	F	29	Housewife.	3 yr.	1	Dust, feathers, tobacco	.....	Improved
29	E. S.	M	31	Chemical worker	1 yr.	1 and 2	Pyrethrum	.....	Changed job
30	J. B.	M	48	Baker.....	9 yr.	1 and 2	Dust, rye, wheat	.....	Fair
31	M. B.	M	32	Dentist.....	2 yr.	1 and 2	All dusts	.....	Improved
32	M. W.	M	46	Furrier.....	10 yr.	1 and 2	Dust, furs	.....	Improved
33	F. W.	M	56	Furrier.....	3 mo.	1 and 2	Dust, furs	.....	Improved
34	M. M.	M	40	Baker.....	5 yr.	1 and 2	Dust, wheat	.....	Improved
35	L. F.	M	31	Paint business	3 mo.	1 and 2	Dust, odors	.....	Fair

* Group 1 was comprised of patients sensitive to general inhalants; group 2, of patients sensitive to occupational inhalants; group 3, of patients sensitive to ingested foods, and group 4, of patients whose vasomotor rhinitis was idiopathic.

TABLE 3.—*Analysis of Fifty-Two Cases of Vasomotor Rhinitis—Continued*

Patient	Name	Sex	Age	Occupation	Duration of Symptoms	Group*	Allergens		Condition at Time of Report
							Inhalants	Foods	
36	G. Z.	F	39	Underwear worker	2 yr.	1 and 3	Dust	Rye	Fair
37	L. G.	M	23	Florist.....	3 mo.	1	Dust	.....	Improved
38	D. G.	F	53	Housewife.	1 yr.	1	Dust	.....	Relieved
39	H. G.	M	40	Butcher....	3 yr.	1 and 2	Dust, feathers	.....	Unimproved
40	S. C.	M	54	.....	2 yr.	4	Idiopathic	.....	Referred
41	C. K.	F	26	Clerk.....	3 mo.	1 and 3	Dust	Grapefruit, potatoes	Improved
42	N. K.	F	15	.....	3 yr.	1	Dust, pollen	.....	Improved
43	M. B.	M	5	.....	1 yr.	1	Dust, pollen	.....	Improved
44	G. H.	F	16	.....	2 yr.	1	Dust, orris	.....	Improved
45	S. K.	M	7	.....	6 mo.	1	Dust, pollen	.....	Improving
46	R. H.	F	26	.....	1 yr.	1 and 3	Dust, tobacco, orris	Cauliflower	Improved
47	L. K.	F	55	Housewife.	5 yr.	1	Dust, tobacco, orris	.....	Improved
48	F. H.	M	30	Stationer..	5 yr.	1 and 2	Dust	.....	Improved
49	C. C.	M	43	Clothing worker	3 yr.	1 and 2	Dust	.....	Improving
50	M. G.	F	25	Clothing worker	1 yr.	1 and 2	Dust, pollen, cotton, kapok	.....	Improved
51	H. W.	F	43	Housewife.	7 yr.	1	Dust, orris, feathers	.....	Relieved
52	G. L.	F	24	Clothing worker	1 yr.	1, 2 and 3	Dust	Grapefruit	Relieved

(*j*) Poultry handlers. Farmers, dealers and butchers are subject to sensitization by the feathers and epitheliums of chickens and other poultry and even by the linseed meal in chicken feed. Hyposensitization frequently proves effective.

(*k*) Bakers. The flours of wheat, rye, buckwheat and other grains are the responsible allergens in this group. In some cases treatment with the specific grain may enable the patient to keep on in the same work, but in others a change of occupation may be required.

(*l*) Paint workers. The fumes, particularly of turpentine, in this occupation often act as the causative irritant. One of the patients was found to be allergic specifically to dust and also to the odor of paint.

Group 3 (patients sensitized by ingested foods).—A large number of the patients in the present series were found to be sensitive to certain foods. Cutaneous tests and elimination diets incriminated the following as etiologic:

(*a*) Milk. A man of 50 years had never suspected that he might be sensitive to milk, and some difficulty was encountered in discovering the cause of his symptoms. Whey and casein in weak dilutions proved unable to excite cutaneous reactions, but further attempts with these components in increasingly stronger concentrations finally produced a moderate reaction to whey. With this as a clue, an exhaustive study of his dietary was made and the conclusion reached that milk and cream

were responsible for the rhinitis. On elimination of these articles from his diet, the symptoms promptly cleared up.

Another patient was himself a rhinologist. Having originally propounded the theory of injecting sodium morrhuate into the inferior turbinates for vasomotor rhinitis, he applied this treatment, among others, to himself. Unable to secure relief, he placed himself in my hands. After intensive cutaneous testing, I was able to obtain a positive reaction only with whey. A marked reaction resulted from the use of 0.001 mg. of total nitrogen of this milk product. Believing this finding to have clinical significance, I advised elimination of milk from his diet, which resulted in immediate cessation of symptoms.

(*b*) Eggs. Sensitivity to eggs encountered in a fairly large number of cases.

(*c*) Wheat. There was a moderately large group of cases of vasomotor rhinitis in which sensitivity to wheat was found.

(*d*) Spices. Ingestion of onion, pepper, mustard, allspice, vinegar, tomato catsup and similar condiments was found to be responsible for the rhinitis in a large number of patients.

(*e*) Meats. Sensitivity to lamb, chicken, veal and red meats was sometimes etiologic.

(*f*) Fish. Both fresh and salt water fish produced symptoms in a large group.

(*g*) Vegetables. Only a few patients exhibited sensitivity to vegetables, and asparagus, tomatoes, onions and potatoes were found to be the offenders.

(*h*) Fruits. Sensitivity to fruit was responsible for many cases. The irritants were grapefruit, pineapple, banana, melon and berries of various kinds.

(*i*) Miscellaneous foods. Among other foods to which an occasional patient was found to be allergic were chocolate and vanilla.

Group 4 (patients whose rhinitis was idiopathic).—This group comprises those who failed to show a cutaneous reaction to any inhalant discoverable from their history and in whom no symptoms of food allergy could be found either by cutaneous tests or by elimination diets or by any other method of study. These were the patients that had to be referred to the rhinologist for diagnosis and for specific and nonspecific methods of treatment.

The cases of 52 patients who are representative of the four groups described have been analyzed in table 3.

COMMENT

Analysis of the cases of the 52 representative patients with perennial rhinitis reveals that in 46, or 89 per cent, the rhinitis was of allergic

origin. The patients were for the most part adults, ranging in age from 20 to 55 years. Two were children of 5 and 7, respectively, and 2 were adolescents of 15 and 16. More males than females were affected, in a ratio of about 2.5 to 1. Symptoms had persisted for periods ranging from three months to sixteen years.

Multiple sensitivities were present in the vast majority of the patients, but with few exceptions rhinitis was the sole clinical manifestation. Hay fever, asthma and urticaria coexisted with the perennial rhinitis in a small percentage.

Inhalant allergens were most often responsible for the rhinitis, but food sensitivity could be demonstrated in 11, or about 24 per cent, as either a primary or a secondary etiologic factor.

Dust sensitivity predominated over all others in frequency, having been present in 44, or 95 per cent, of the patients. Sensitivity to occupational allergens other than dust was demonstrated in 19, or 41 per cent, as follows: 6 clothing workers, 5 furriers, 4 bakers, 1 feather worker, 1 chemical worker, 1 paint worker and 1 butcher. The specific occupational allergens, besides dust, comprised animal fur (or dyes and chemicals used by furriers), feathers, wool, cotton, kapok, wheat, rye, pyrethrum and the odor of paint. One florist, who naturally would be suspected of allergy to plants or pollens, proved sensitive only to dust.

Housewives composed 13 per cent of the allergic series. They were sensitive to dust, orris root, feathers, tobacco, perfumes and animal epitheliums. One housewife had symptoms only on Mondays, while gathering up soiled clothing for the laundry.

The 4 patients 16 years of age and less were allergic to dust, pollen and orris root.

Treatment consisted of injecting or avoiding all the specific allergens that could be demonstrated, and for some patients with intractable rhinitis both measures were necessary. When asthma also was present, it cleared up under treatment earlier than did the nasal symptoms.

The methods that proved successful in the practical management of allergic vasomotor rhinitis in a large series of cases have been outlined on page 4. The malady is not easy to bring to a satisfactory conclusion, as one often encounters extreme difficulty in identifying the causative allergens. In some instances, these are of a nonspecific character, like vapors, smoke, fumes or heat, cold, light and so on. Information regarding such apparently irrelevant factors is not readily elicited from the patient, and even when found to be causative they cannot be used for desensitization therapy. The only possible treatment is avoidance, which in many cases means that the patient must give up his job. Moreover, even if the allergens are specific, it may be that they are met with only casually, and for this reason are overlooked by both the patient and the physician. Never-

theless, the cumulative effect of the casual contact irritants may be the actual cause of the symptoms. The same holds true of multiple sensitivities, no one of which may be sufficient to give a marked cutaneous reaction to the specific allergen, yet the sum total is what gives rise to the rhinitis. Then again, a suspected food may fail to produce a positive cutaneous reaction and still be the cause of clinical symptoms. Cutaneous testing for food allergy in adults cannot be relied on but is useful as a supplement to other diagnostic measures, such as elimination diets.

The case history must reveal practically every detail of the patient's routine. If the causation is obscure, the home survey nurse may be needed to check on the sports, hobbies and social and home contacts, which frequently are forgotten or disregarded by the patient himself. It is only by such combined studies of all possible factors that one may hope to attain any measure of success in the management of this type of vasomotor rhinitis.

Since many cases arise from conditions in industry, it is important to obtain a detailed account of the materials and even the fumes and odors encountered by the patient in his work. In my experience with furriers, for example, avoidance of some fur that produced positive cutaneous reactions did not always relieve the symptoms, and the conclusion was that the fumes of dyes and chemicals used in the work were the real cause. The chemicals capable of causing allergic reactions in furriers are: (1) paraphenylenediamine, otherwise known as ursol D, paraaminophenol (ursol P), and a number of other ursols used for dyeing furs (Forker [7] recorded positive scratch tests in allergic patients with ursol D, DD [gray-white], DF [black-gray], SB [black-blue-green], DMG [black], P [dark gray–black] and NZ [light gray–blue]; (2) insecticides with which the skins are treated, notably paradichlorobenzene; (3) chinondiimin, which results from incomplete oxidation of ursol by the hydrogen peroxide with which it is washed. As in the case of other chemicals, however, a positive cutaneous reaction seldom can be elicited with these, and desensitization cannot be accomplished. Complete or partial removal of the patient from his work offers the only hope of relief in such cases.

Eyermann [8] studied 181 patients with vasomotor rhinitis due to ingested foods and found that the foods most frequently eaten were the ones most often responsible for sensitization. He concluded that multiple cutaneous and clinical sensitivity was the rule with these patients rather than the exception. In his cases of perennial nasal

7. Forker, H.: Ueber die biologisch verschiedene Wirksamkeit der einzelnen Ursole, Arch. f. Gewerbepath. u. Gewerbehyg. **6**:275 (Dec.) 1935.

8. Eyermann, C. H.: Food Allergy as a Cause of Vasomotor Rhinitis, South. M. J. **31**:210 (Feb.) 1938.

allergy associated with hay fever, the avoidance of food allergens and inhalants other than pollen usually cleared up the condition. He therefore advised that the influence of foods and inhalants be investigated before resorting to injections of pollen. My own experience with patients of this type has led me to the same conclusion.

Spain [9] found cases of perennial coryza caused by ingested food and drugs to be rare as compared with those due to inhalants. He mentioned milk, eggs, cereals, fish, shellfish, nuts, mustard and chocolate as causative factors; and among the drugs, acetylsalicylic acid, quinine, methyl salicylate (oil of wintergreen) and tincture of larkspur.

While it was not possible to find any instance of allergy due to a fungus in the present series, fungi have been reported as allergens so often by others that they merit careful consideration in making a diagnosis of vasomotor rhinitis. Feinberg,[10] Feinberg and Little,[11] Chobot, Dundy and Schaffer,[12] Harris [13] and others have demonstrated sensitivity to Alternaria, Aspergillus, Mucor, Pencillium and numerous other species of fungi, including yeasts, molds, rusts and smuts, which caused among other allergic manifestations seasonal and perennial rhinitis.

SUMMARY AND CONCLUSIONS

The management of uncomplicated nonseasonal allergic vasomotor rhinitis has been described, with special emphasis on exhaustive methods of diagnosis and intensive treatment.

The condition is characterized by blocking of the nasal passages, with persistent rhinorrhea and, in adults, violent and prolonged paroxysms of sneezing. The nasal mucosa is swollen and boggy, with a pale pinkish gray color, and the secretions usually exhibit marked eosinophilia.

In many cases the causes are obscure, and a detailed investigation of the patient's background, as well as exhaustive testing, is necessary in order to identify the responsible allergens.

A limited series of 52 cases has been analyzed, in which twenty-two different substances were incriminated. Most of these were inhalants,

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9. Spain, W. C.: Diagnosis and Treatment of Atopic Coryza (Perennial Hay Fever), Ann. Otol., Rhin. & Laryng. **34**:1089 (Dec.) 1925.

10. Feinberg, S. M.: Seasonal Hay Fever and Asthma Due to Molds, J. A. M. A. **107**:1861 (Dec. 5) 1936; Asthma and Allergic Rhinitis from Molds; Analysis of Ninety Cases, Journal-Lancet **57**:87 (March) 1937.

11. Feinberg, S. M., and Little, H. T.: Studies in the Relation of Microörganisms to Allergy: II. Role of Yeasts in Allergy (Preliminary Report), J. Allergy **6**:564 (Sept.) 1935.

12. Chobot, R.; Dundy, H., and Schaffer, N.: Relationship of Mold Reactions to Clinical Symptoms, J. Allergy **12**:46 (Nov.) 1940.

13. Harris, L. H.: Allergy to Grain Dusts and Smuts, J. Allergy **10**:327 (May) 1939; Respiratory Mold Allergy, ibid. **12**:279 (March) 1941.

and a large proportion were met with in the patients' occupations. Food allergy accounted for a small percentage of the cases.

Other investigators have identified molds, yeasts, rusts and smuts, insect emanations and a variety of other inhalants as etiologic factors in perennial vasomotor rhinitis. Ingested drugs and chemicals also have been implicated.

In many of the patients the rhinitis is of long standing and stubbornly resistant to treatment. These require a special technic of intensive therapy, which has been described.

235 East Twenty-Second Street.

CERTAIN ALIPHATIC COMPOUNDS AS NASAL VASOCONSTRICTORS

ARTHUR W. PROETZ, M.D.
ST. LOUIS

Probably the most important single group of drugs available to the rhinologist are the vasoconstrictors.

Cocaine, originally used in the nose by Jelinek [1] in 1884, and epinephrine, first employed intranasally by Velich and Königstein [1] in 1897 and first introduced into this country by Swain [2] in a paper read before this association in 1898, headed a long procession of vasoconstrictors, which differed largely in their potency and their side effects. This procession is still on its way.

The two closely related substances which are the subject of this paper have the distinction of being the first to emanate from the relatively simple aliphatic group of compounds. It was shown only last year by Shonle and Rohrmann [3] that certain aliphatic amines having the amino group attached to the second carbon atom exhibit pressor action. This led Chen and Swanson [4] to investigate them thoroughly on a pharmacologic basis. Chen will be remembered as having in 1925 contributed largely to the standardization and scientific knowledge of the ancient drug ephedrine, which to the best of my information was made known to rhinologists by Fetterolf and Sponsler [5] a year later.

These substances also are not new, having been recognized as early as 1863. Until now, however, they have been merely chemical curiosities.

2-AMINOHEPTANE

The first is heptane into which a single amino group has been introduced in the *2* position (fig. 1). It is remarkable that the compounds

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From the Department of Otolaryngology, Washington University School of Medicine.

Read at the Sixty-Fourth Annual Meeting of the American Laryngological Association, Atlantic City, N. J., May 26, 1942.

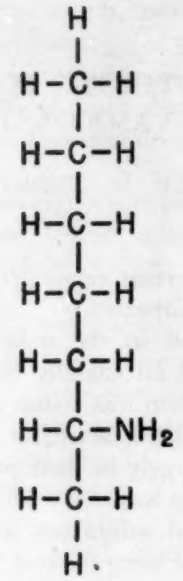
1. Cited by Wright, J.: A History of Laryngology and Rhinology, Philadelphia, Lea & Febiger, 1914.

2. Swain, H. L.: The Local Use of the Aqueous Extract of the Suprarenal Glands of the Sheep in the Nose and Throat, Tr. Am. Laryng. A., 1898, p. 165.

3. Shonle, H. A., and Rohrmann, E.: Personal communication to the author, to be published.

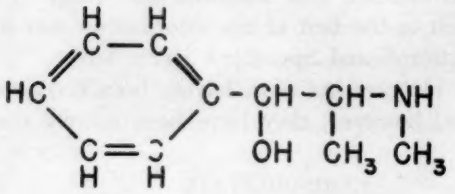
4. Chen, K. K., and Swanson, E. E.: To be published.

5. Fetterolf, G., and Sponsler, M. B.: Ephedrine Sulphate, the Alkaloid of Ma Huang, Arch. Otolaryng. **2**:132 (Aug.) 1925.

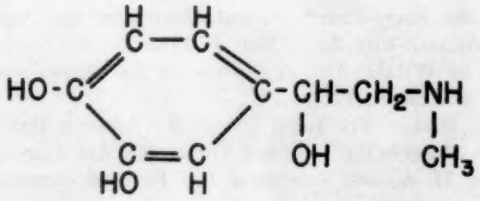


$C_7H_{15}NH_2$

Fig. 1.—Structural formula for 2-aminoheptane.



Ephedrine

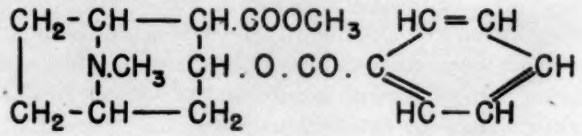


Epinephrine

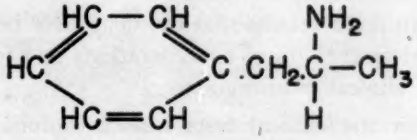
Fig. 2.—Structural formulas for ephedrine and epinephrine.

bearing this group in the 1 or the 3 position are relatively inert. Not only is 2-aminoheptane a very potent vasoconstrictor, but it is free from some of the undesirable local and systemic side effects which characterize cocaine, epinephrine, amphetamine and ephedrine, as will be shown. Chemically it is much simpler than any of these (figs. 2 and 3).

Because of its greater solubility, the sulfate is employed instead of the alkaloid itself. I shall briefly summarize the pharmacologic characteristics of this compound as developed by Chen and his associates, and its effects on ciliated epithelium and the tissues of the human nose as reported by me:[6] 2-aminoheptane sulfate injected into animals raises the blood pressure; it decreases the volume of the nasal erectile bodies; it dilates the pupils of rabbits on local application.



COCAINE



AMPHETAMINE

Fig. 3.—Structural formulas for cocaine and amphetamine.

Its activity and toxicity as compared with ephedrine and amphetamine are as follows:

Activity (in cats)

Drug	Activity, Percentage
Ephedrine sulfate	100
Amphetamine sulfate	66
2-aminoheptane sulfate	52

Toxicity (injected intravenously in mice)

Drug	Median Lethal Dose (M [L $D_{50} \pm$ S.E.])
Ephedrine sulfate	109.8 ± 5.86
2-aminoheptane sulfate	36.93 ± 1.335
Amphetamine sulfate	5.603 ± 0.427

6. Proetz, A. W.: 2-Amino-Heptane Sulfate as a Nasal Vasoconstrictor, Ann. Otol. Rhin. & Laryng. **51**:112 (March) 1942.

Twenty-five milligrams administered by mouth to each of 5 subjects failed to produce any increase in blood pressure.

The p_H of a 2 per cent solution has been determined to be 5.7.

An isotonic solution of this strength may be prepared as follows: 2-aminoheptane sulfate, 2 Gm.; sodium chloride, 0.406 Gm.; distilled water, to make 100 cc.

Effect on Ciliated Epithelium of Rabbits.—Fragments of ciliated epithelium were removed from the nasal chambers of rabbits killed by injection of air and were immersed in solutions of 2-aminoheptane sulfate of various strengths. Hypotonic solutions were rendered isotonic by the addition of proper amounts of sodium chloride.

Specimens immersed continuously in 2 per cent solution remained active for periods from forty-six minutes to two hours and fifteen minutes.

Specimens immersed continuously in 4 per cent solution remained active for periods from forty-five to more than sixty-five minutes.

A specimen immersed continuously in 1 per cent solution in distilled water remained active for twenty minutes.

A specimen immersed continuously in 2 per cent solution in distilled water remained active for seventy-eight minutes.

A specimen immersed continuously in 4 per cent solution in distilled water remained active for fifty-four minutes.

It is concluded from these results that the drug may be applied with impunity to the ciliated epithelium in concentrations and for periods far in excess of the usual clinical requirements.

Clinical Tests.—For the clinical tests isotonic solutions were prepared by adding the requisite amount of sodium chloride to aqueous solutions of 2-aminoheptane sulfate. They were applied to the nasal mucosa of patients in concentrations of 0.5, 1 and 2 per cent. Comparison was also made between the effects of these concentrations and those of a 2 per cent solution of ephedrine sulfate made isotonic with sodium chloride. A series of 119 such applications yielded the following information:

With the 2 per cent solution there were complete shrinking and pronounced pallor in 21 of 31 observations, satisfactory shrinking in 5 and little response to either 2-aminoheptane sulfate or ephedrine sulfate in 5. In all cases the ischemia exceeded that produced by 2 per cent ephedrine sulfate.

With the 1 per cent solution (31 observations) shrinking was still uniformly greater than that produced by 2 per cent ephedrine sulfate.

With the 0.5 per cent solution (57 observations) it could be said that on the whole the shrinking approximated that produced with 2 per cent solution of ephedrine sulfate.

Pallor due to ischemia was not noted when the concentration was less than 1 per cent.

It will be seen therefore that from a clinical standpoint 2-aminoheptane sulfate compares favorably with ephedrine sulfate as a vasoconstrictor. To my mind, however, its principal advantage lies in its apparent freedom from local and general side effects and especially in its prolonged action. The effect following the use of the stronger solutions

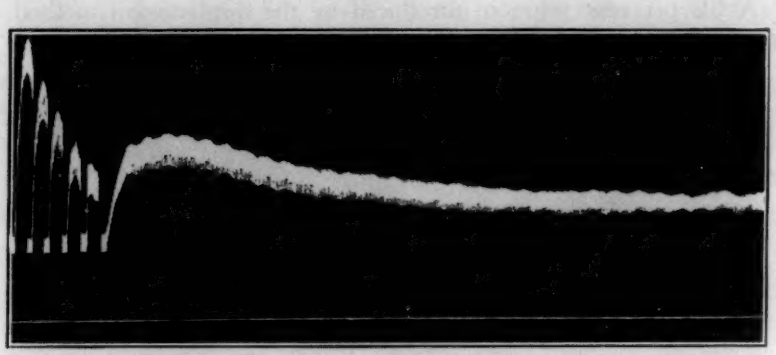


Fig. 4.—Action of 2-aminoheptane on blood pressure.

A male cat, weighing 2.56 Kg., was decerebrated and pithed. Artificial respiration was furnished, and the blood pressure in the left carotid artery was recorded.

The first 5 tracings, reading from left to right, were obtained after intravenous injections of solutions of epinephrine hydrochloride containing 10, 8, 6, 4 and 3 micrograms, respectively. The last curve, obtained with 1 mg. of 2-aminoheptane sulfate, also given by vein, is equivalent in height to that recorded with about 4 micrograms of epinephrine hydrochloride.

This and the following tracings were made by Dr. K. K. Chen, of the Lilly Research Laboratories, and are reproduced with his permission.

Fig. 5.—Effect of 2-aminoheptane on nasal volume.

A male cat, weighing 2.575 Kg., was anesthetized with sodium amytal injected intraperitoneally in a dose of 70 mg. per kilogram of body weight.

The first 5 tracings, reading from left to right, show the quantitative responses to intravenous injections of epinephrine hydrochloride in doses of 10, 6, 4, 2 and 1 micrograms. The last tracing, recorded after 0.5 mg. of 2-aminoheptane sulfate was injected intravenously, is equivalent in height to that obtained with 2 micrograms of epinephrine hydrochloride. In other words, the results in this experiment also indicate that 1 mg. of 2-aminoheptane sulfate has the same intensity of action as 4 micrograms of epinephrine hydrochloride (fig. 4).

often persisted throughout the day. Patients were conscious of the difference and mentioned it. None of the 119 patients complained of local irritation, systemic reactions or subsequent nasal obstruction.

The tracings in figures 4, 5 and 6, kindly supplied by Dr. K. K. Chen, of the Lilly Research Laboratories, show graphically the characteristics of the action of this drug.

I have used 2-aminoheptane sulfate continuously in my practice for the past ten months in concentrations from 0.5 to 2 per cent. During this time it has produced no ill effects in any case.

A 0.2 per cent solution introduced by the displacement method is effective and well tolerated.

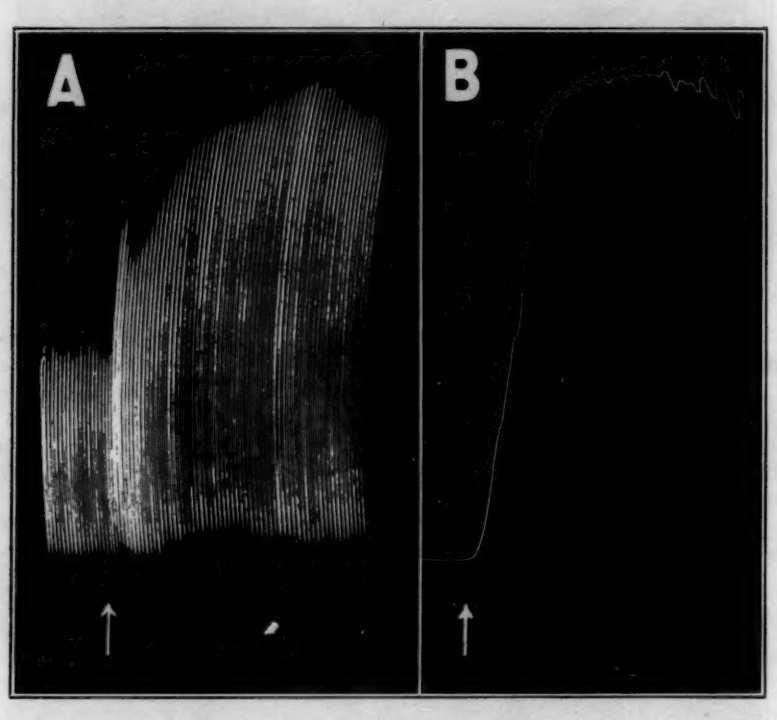


Fig. 6.—Action of 2-aminoheptane on smooth muscle.

(*A*) A strip of isolated rabbit intestine was suspended in Tyrode's solution at a temperature of 38 C. At the arrow, 0.3 cc. of a twentieth-molar solution of 2-aminoheptane sulfate was added, making a concentration of about 1:20,000. Note the augmentation of peristaltic movements.

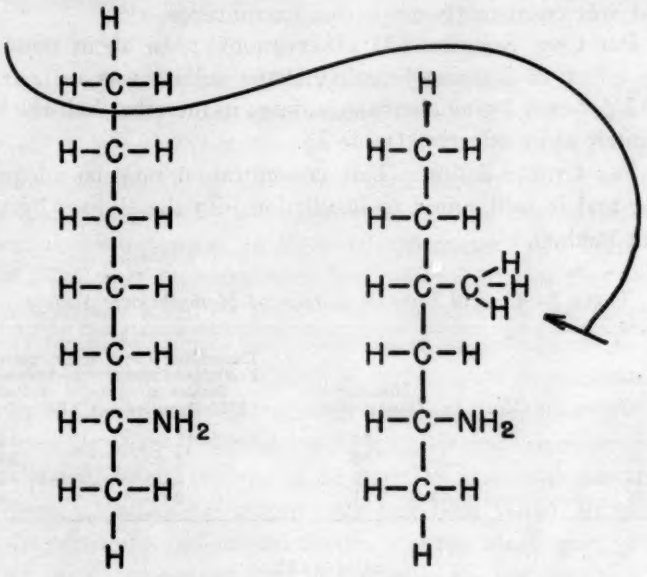
(*B*) A strip of guinea pig uterus was immersed in Locke's solution at 38 C. At the arrow, the same amount of 2-aminoheptane sulfate was applied. Note the spastic contraction.

2-AMINO-4-METHYLHEXANE SULFATE

These results naturally aroused speculation as to the possible effects of other combinations of the radicals commonly encountered in the constrictor groups with the heptane base in the place of the benzene ring.

The investigators at the Lilly Research Laboratories have prepared several such combinations. At the present writing one other compound, 2-amino-4-methylhexane (fig. 7), gives promise of usefulness.

Laboratory Determinations.—Again with the sulfate, experiments similar to those with 2-aminoheptane were conducted. The ciliated epithelium from the rabbit's nose was even more tolerant of this drug than the first, as is shown in table 1.



$$C_7H_{15}NH_2$$
$$= C_6H_{12}CH_3 \cdot NH_2$$

Fig. 7.—Structural formula for 2-amino-4-methylhexane sulfate.

TABLE 1.—*Experimental Data on 2-Amino-4-Methylhexane Sulfate* *

Solution, %	Ciliary Activity Persisted
2.0	2 hr. 25 min. to 4 hr. 6 min.
1.0	3 hr. 28 min. to 4 hr. 22 min.
0.5	2 hr. 46 min. to 4 hr. 9 min.

* The solutions were made isotonic with sodium chloride. Specimens immersed for two minute periods in solutions of increasing concentrations beginning at 1 per cent remained active until subjected to concentrations of 6 and 8 per cent.

Clinical Tests.—Clinical tests were made with the following concentrations of 2-amino-4-methylhexane sulfate rendered isotonic with sodium chloride:

0.5 Per Cent Solution (30 observations): In 13 cases shrinking exceeded that with 0.5 per cent solution of 2-aminoheptane sulfate.

Constriction was extreme, and pallor was prominent. The action here appeared greater than that required for any clinical effect, excepting perhaps hemostasis.

0.25 Per Cent Solution (32 observations): Constriction with only 3 exceptions equaled that with 0.5 per cent 2-aminoheptane sulfate. It still exceeded the usual requirements.

0.12 Per Cent Solution (36 observations): The effect of this solution also was exceeded by that of the 2-aminoheptane sulfate on only 3 occasions and was consistently ample for examination.

0.06 Per Cent Solution (31 observations): In about one-half the tests the effect of 2-amino-4-methylhexane sulfate was comparable to that of 0.5 per cent 2-aminoheptane sulfate; in the other half the heptane was definitely more effective (table 2).

0.06 Per Cent Solution: This concentration may be adequate for home use and is well suited to instillation into the sinuses by the displacement method.

TABLE 2.—*Clinical Data on 2-Amino-4-Methylhexane Sulfate*

Solution, per Cent	Number of Observations	Exceeding 0.5% 2-Aminoheptane Sulfate in Effectiveness	Approximating 0.5% 2-Aminoheptane Sulfate in Effectiveness
0.5	30	13	16
0.25	32	1	28
0.12	36	1	32
0.06	31	0	18

SUMMARY

Immersion of detached specimens of nasal ciliated epithelium (rabbit) in solutions of 2-aminoheptane sulfate and 2-amino-4-methylhexane sulfate for varying periods indicates that these drugs are not deleterious to ciliary activity as applied under clinical conditions.

Clinical tests embracing 264 observations indicate that both drugs are effective nasal vasoconstrictors; that 0.5 per cent solution of 2-aminoheptane sulfate approximates in its clinical potency 2 per cent solution of ephedrine sulfate and that 0.12 per cent solution of 2-amino-4-methylhexane sulfate approximates in its clinical potency 0.5 per cent solution of 2-aminoheptane sulfate.

A 0.2 per cent solution of either drug introduced into the sinuses by the displacement method is effective and well tolerated.

Absorption of these drugs by the nasal mucosa is slow; the effect is prolonged, and rise in blood pressure does not occur after nasal application or oral ingestion. No other systemic effect or after-effect has been observed.

3720 Washington Boulevard.

TENSION PNEUMOTHORAX AND MEDIASTINAL EMPHYSEMA AFTER TRACHEOTOMY

GENERAL STUDY, WITH ANALYSIS OF SEVENTEEN CASES IN A SERIES OF ONE HUNDRED AND TWENTY-SIX TRACHEOTOMIES FOR ACUTE OBSTRUCTIVE INFECTIONS OF THE LARYNX, TRACHEA AND BRONCHI DURING THE PAST DECADE

MAJOR A. HARRY NEFFSON

MEDICAL CORPS, AIR FORCES, ARMY OF THE UNITED STATES

Tension pneumothorax and mediastinal emphysema occur more frequently than a review of the literature would lead one to believe. The fact that so few instances of these complications have been reported does not mean that they are rare, but rather indicates that they often go unrecognized. Even on postmortem examination the correct diagnosis may be missed unless the possibility of pneumothorax is borne in mind and the proper technic is used to demonstrate its presence.

In 1884 Champneys[1] stated that in an examination of the postmortem records of the Hospital for Sick Children (London) from 1860 to 1882 he had found reports of 82 cases of pneumothorax following tracheotomy. Mediastinal emphysema had been noted, in passing, in 5 cases (6 per cent), but pneumothorax was not mentioned. However, in the two years that elapsed after he published his first paper on mediastinal emphysema and pneumothorax, in 1882, there were 28 cases in which postmortem examination was done after tracheotomy and of these mediastinal emphysema was noted in 16 cases, including 2 with pneumothorax. In all cases the examination had been performed under water.

Figi,[2] reporting a series of 200 consecutive cases of tracheotomy performed at the Mayo Clinic, did not mention the occurrence of either pneumothorax or mediastinal emphysema. In 34 per cent of his cases tracheotomy was urgent because of impending asphyxia. However, in the large majority the obstruction was due to laryngeal carcinoma, and

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From the Willard Parker Hospital.

1. Champneys, F. H.: Artificial Respiration in Stillborn Children—Mediastinal Emphysema and Pneumothorax in Connection with Tracheotomy: An Experimental Inquiry, Med.-Chir. Tr., London (*a*) **65**:75, 1882; (*b*) **67**:101, 1884.

2. Figi, F. A.: Tracheotomy: A Study of Two Hundred Cases, Ann. Otol., Rhin. & Laryng. **43**:178 (March) 1934.

only 17 patients were under the age of 5 years. In their book on the larynx and its diseases, the Jacksons,[3] discussing the complications of tracheotomy, dismissed the entire subject with the following statement:

> . . . mediastinal emphysema may develop from working of the air along between the tissue layers under the deep fascia, aided by the normal negative pressure at inspiration; it may even reach the pleural cavity causing pneumothorax. If double, oxygen should be insufflated to prevent asphyxia.

Pneumothorax complicating tracheotomy has been reported by Wilks and Moxon,[4] Champneys,[1] Iglauer,[5] Myerson,[6] Wiethe,[7] Neffson and Wishik,[8] Simpson,[9] Neffson and Bullowa,[10] Michels [11] and Dolgopol and Stern.[12] Leiner [13] cited 9 cases of mediastinal emphysema following tracheotomy in children; Graebner [14] reported 4 cases of pneumomediastinum following tracheotomy for acute laryngeal obstruction and 1 case in which the condition occurred spontaneously in a patient with acute laryngeal obstruction.

ANALYSIS OF MATERIAL

In reviewing a series of 126 consecutive cases of tracheotomy performed during the past decade at the Willard Park Hospital to relieve

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3. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937.

4. Wilks, S., and Moxon, W.: Lectures on Pathological Anatomy, ed. 2, London, J. & A. Churchill, 1875, p. 308.

5. Iglauer, S.: Accidental Pneumothorax During Tracheotomy, Ann. Otol., Rhin. & Laryng. **24**:303, 1915.

6. Myerson, M. C.: Perforation of the Bronchus with Notes on Bronchoscopic Anatomy, Ann. Otol., Rhin. & Laryng. **41**:1203 (Dec.) 1932.

7. Wiethe, C.: Ueber Pneumothorax bei Tracheotomia inferior, Monatschr. f. Ohrenh. **67**:57 (Jan.) 1933.

8. Neffson, A. H., and Wishik, S. M.: Acute Infectious Croup: A General Study of Acute Obstructive Infections of the Larynx, Trachea and Bronchi with an Analysis of Seven Hundred and Twenty-Seven Cases, J. Pediat. **5**: (*a*) 433 (Oct.); (*b*) 617 (Nov.); (*c*) 776 (Dec.) 1934.

9. Simpson, W. L.: Pneumothorax Complicating Tracheotomy in Fulminating Laryngotracheobronchitis, Arch. Otolaryng. **26**:411 (Oct.) 1937.

10. Neffson, A. H., and Bullowa, J. G. M.: Influenza with Simultaneous Bilateral Spontaneous Pneumothorax and Subcutaneous Emphysema: Report of a Case with Comment on the Mechanism of Production, Arch. Otolaryng. **28**:388 (Sept.) 1938.

11. Michels, M. W.: Pneumothorax and Mediastinal Emphysema Complicating Tracheotomy, Arch. Otolaryng. **29**:842 (May) 1939.

12. Dolgopol, V. B., and Stern, M. E.: Interstitial Emphysema of the Lung with Spontaneous Pneumothorax and Subcutaneous Emphysema—Demonstration of Air in the Septums of a Human Lung, Arch. Otolaryng. **31**:140 (Jan.) 1940.

13. Leiner, C.: Mediastinales Emphysem bei tracheotomierten Kindern, Jahrb. f. Kinderh. **58**:448, 1903.

14. Graebner, H.: Pneumopericardium and Pneumomediastinum in Cases of Acute Obstructive Laryngitis, Arch. Otolaryng. **29**:446 (March) 1939.

acute nondiphtheritic obstructive infections of the larynx, trachea and bronchi, 17 cases of pneumothorax, an incidence of 13.5 per cent, were encountered.[15] In the first five years, out of 32 cases of tracheotomy there were 8 cases of pneumothorax, an incidence of 25 per cent. In the last five years, out of 94 cases of tracheotomy there were 9 cases of pneumothorax, an incidence of 10 per cent. In the last three years, out of 74 cases of tracheotomy there were only 6 cases of pneumothorax, an incidence of only 8 per cent. Thus, with an increase in the relative frequency of tracheotomy, from 4.3 per cent in the first five years to 19.4 per cent in the last three years, there was a lowering in the incidence of pneumothorax of from 25 to 8 per cent.

In the 17 cases of pneumothorax, there were 12 males and 5 females —the same proportion as that in the general series of tracheotomies. The ages ranged from 6 months to 4 years, the most frequent age incidence being from 1 to 2 years.

In 8 cases the pneumothorax was unilateral and in 9 bilateral. Of the 8 cases of unilateral pneumothorax, 2 had a fatal termination, a mortality rate of 25 per cent. Of the 9 cases of bilateral pneumothorax, 8 ended fatally, a mortality rate of 89 per cent. Of the 8 cases of unilateral pneumothorax, the left side was involved in 5 and the right in 3.

In 93 of the 126 cases of tracheotomy, intubation had preceded the operation. Pneumothorax occurred in 6 of the 33 cases of tracheotomy without intubation, or in 18 per cent, and in 11 of the 93 cases of tracheotomy with previous intubation, or in 12 per cent. Of the 6 cases in which intubation did not precede tracheotomy, death occurred in 4 (mortality rate 67 per cent), and of the 11 cases in which intubation was done previously, the termination was fatal in 6 (mortality rate 55 per cent). In the 11 cases in which intubation was done prior to tracheotomy, from 2 to 20 intubations were done, an average of 6.8, over a period of one to seventeen days, the average period being six days.

The pneumothorax was noted within a few hours after operation in 9 cases. In 2 cases signs of pneumothorax or mediastinal emphysema were noted during operation, before the trachea was incised. In these there was ballooning of the pleura, unaccompanied by lung tissue, into the tracheotomy wound with each expiration. (This phenomenon was reported by Neffson and Bullowa[10] and was also mentioned by Michels.[11]) In 1 case death occurred during tracheotomy and pneumothorax was observed post mortem. In the remaining 5 cases the pneumothorax was first noted from one to three days after operation and lasted from three to twelve days.

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15. Neffson, A. H.: Tracheotomy in Cases of Acute Obstructive Nondiphtheritic Infections of Larynx, Trachea and Bronchi: General Study, with an Analysis of One Hundred and Twenty-Six Consecutive Cases Occurring During the Past Decade, Arch. Otolaryng. **36**:773 (Dec.) 1942.

In 10 of the 17 cases in which pneumothorax developed, tracheotomy was performed either on admission or within the first twenty-four hours; of these, death occurred in 8—a mortality rate of 80 per cent. In 7 cases tracheotomy was done from two to twenty-one days after administration; in 2 of these the patients died, a mortality rate of 28 per cent. Of the 10 fatal cases, death occurred within twenty-four hours after operation in 6 and from two to ten days after operation in 4. The respective causes of death in the 4 cases in which death occurred from two to ten days after operation were as follows: influenzal meningitis, endocarditis, mediastinitis and empyema, case 1; asphyxia from a pretracheal abscess, which ruptured and flooded the trachea, case 2; acute laryngotracheobronchitis with thick asphyxiating secretion and pneumothorax, case 3, and sudden death from no apparent cause, probably cardiac, with no postmortem examination, case 4. In the first 2 cases pneumothorax was not present at the time of death.

Postmortem examination was made in 8 of the 10 cases. In 3 of these death had occurred from three to ten days after tracheotomy, and the postmortem studies did not reveal anything of significance in explaining the cause of the pneumothorax. In the other 5 cases death occurred within forty-eight hours after tracheotomy, and the significant postmortem observations were as follows:

Case 1: Death in twenty hours. Bilateral tension pneumothorax; extensive mediastinal blebs, but no gross rupture seen. No marginal pleural emphysema or interstitial emphysema. Pleural domes intact.

Case 2: Death in five hours. Bilateral tension pneumothorax; large mediastinal bullae, each having to be punctured individually; no gross rupture seen. No pleural blebs. Pleural domes intact.

Case 3: Death on operating table before trachea was incised. Tension pneumothorax on right side; bullous emphysema of pleura of right lung; no rupture seen. No mediastinal emphysema. Pleural domes intact.

Case 4: Death in forty-eight hours. Bilateral tension pneumothorax; mediastinal emphysema; no gross rupture seen. No interstitial emphysema in the septum; no pleural blebs. Pleural domes intact.

Case 5: Death in twenty-four hours. Tension pneumothorax on left side; interstitial emphysema; no pleural blebs; no mediastinal emphysema. Pleural domes intact.

To these cases may be added another in which tracheotomy was performed at the Mount Sinai Hospital [16] because of sudden asphyxia from acute phlegmonous laryngitis with supraglottic edema. Bilateral tension

16. This case occurred in the service of Dr. Rudolph Kramer and is reported with permission of the pathologic laboratories of the Mount Sinai Hospital.

pneumothorax was noted one hour after tracheotomy. There was a positive pressure of from 6 to 14 cm. of water, and several liters of air was removed during the next eighteen hours, up to the time of death. Postmortem examination showed extensive subcutaneous emphysema, bilateral tension pneumothorax with collapse of the lungs, numerous air blebs over the mediastinum, no demonstrable rupture of the visceral, parietal or mediastinal pleura and no interstitial emphysema.

There were 30 cases of mediastinal emphysema, including the pericardial type. In these the diagnosis was made by roentgenographic and physical signs. They presented no special problem with regard to decompression. In 5 of these cases mediastinitis and empyema were contributing causes of death.

Subcutaneous emphysema was of rather frequent occurrence but presented no special problems and had no influence on the prognosis. The air was absorbed within seven days.

REVIEW OF LITERATURE

The following experimental work, clinical and pathologic material and discussions gathered from the literature are presented in brief because of their help in formulating a conception of the mechanisms in the production of tension pneumothorax and mediastinal emphysema after tracheotomy.

Champneys,[1] in 1882, while testing the value of different methods of artificial respiration for resuscitating stillborn infants, was struck by the occurrence of mediastinal emphysema and pneumothorax in some of his cases. In his experiments, tracheotomy was performed and a cannula was tied into the trachea. This cannula was connected with tubing to a manometer, and different methods of artificial respiration were tested. Mediastinal emphysema occurred in 7 of the 21 bodies used, and pneumothorax developed in 5 of these 7 cases of mediastinal emphysema (1 bilaterally, 3 on the right side and 1 on the left side). He stated:

> Pneumothorax . . . never occurred without emphysema and . . . was probably a later sequel of the emphysema . . . Air was observed escaping from the mediastinum into the pleural sac. . . . It appeared, then, that the air traveled from the tracheotomy wound into the mediastinum, which in some cases it ruptured, producing pneumothorax.[1a]

He explained its occurrence as follows:

> If the entrance of air into the trachea is interfered with during forcible inspiratory efforts and if at the same time the deep cervical fascia is opened as a preliminary to tracheotomy, then a potential passage into the thorax becomes established, and it is through this passage that the air penetrates the mediastinum. If the pressure is equalized it goes no farther, but if the pressure is not equalized it tends to burst the medias-

tinum and distend the pleural sac. Mediastinal emphysema is due to diminished pressure in the thorax during inspiration, in contradistinction to the common form of emphysema of the neck after tracheotomy, which is purely of expiratory origin.

With respect to avoiding mediastinal emphysema and pneumothorax, he concluded:

> Supposing obstruction to exist in the larynx, the period of greatest danger is the interval between the division of the deep cervical fascia and the establishment of efficient patency of the trachea. Elevation of this fascia away from the trachea renders the entry of air beneath it more easy. Bungling in inserting the tube increases the danger by prolonging the dangerous period. Insertion of the tube beneath the deep cervical fascia renders the entrance of air easy above all other conditions. . . . The incision in the deep cervical fascia should not be longer than necessary . . . and it should on no account be raised from the trachea.[1a]

As previously noted, he found postmortem records in 16 instances of mediastinal emphysema, including 2 with pneumothorax, among 28 cases of tracheotomy followed by death at the Hospital for Sick Children. He also collected 3 cases of mediastinal emphysema, including 2 of pneumothorax, from 27 cases in which death followed tracheotomy at St. Bartholomew's Hospital. He referred to Wilks and Moxon,[4] who cited 2 cases of pneumothorax following tracheotomy in which there was extensive mediastinal emphysema and in which they asserted that the air from the mediastinum ruptured through the pleura.

Kelman [17] described pronounced interstitial and mediastinal emphysema in cases of death from influenzal bronchopneumonia. In an attempt to determine the pathology of the emphysema, she performed tracheotomies on rabbits and varied the intrapulmonic pressure. She found pronounced mediastinal and anterior pericardial emphysema, and, by putting saline solution into the chest, she observed air escaping at the root of the lung in the vicinity of the great vessels and the pleural reflection. She concluded:

> First, a vesicular emphysema is produced. Increased intrapulmonary pressure causes rupture of some of the superficial distended air vesicles and there is an escape of air under the visceral pleura. [This then tracks under the pleura to the hilus, instead of breaking through the pleura.] It travels along the pleural reflexions over the pulmonary [vessels] . . . to the mediastinum.

She observed that toxins, particularly those of influenza, caused weakening of the alveoli, with respiratory distress.

Joannides and Tsoulos [18] produced tension pneumothorax in dogs by varying the intrapulmonic pressure. They observed large air blebs

17. Kelman, S. R.: Experimental Emphysema, Arch. Int. Med. **24**:332 (Sept.) 1919.

18. Joannides, M., and Tsoulos, G. D.: Etiology of Interstitial and Mediastinal Emphysema, Arch. Surg. **21**:333 (Aug.) 1930.

near the hili of the lungs at the junction of the visceral and the parietal pleura. They stated that on rupture of these blebs pneumothorax was produced. A high proportion (80 per cent) of cases of air embolism was found in animals in which interstitial emphysema was produced by the intratracheal injection of compressed air. They stated:

> What probably happens is that [with] the increased alveolar tension . . . more air passes into the capillaries than occurs under normal tension, but after the larger capillaries are reached the tension is again changed to normal with a resulting air embolism.

Macklin,[19] working with cats and rabbits, produced pulmonary and mediastinal emphysema and pneumothorax by local overinflation of the lung substance. By fixing the lungs immediately after death he was able to trace the pathway by which the air spread. He concluded:

> The aberrant air . . . enters the vascular sheaths through numerous minute openings in the floors of the alveoli which overlie the finer ramifications of the pulmonary arteries and veins. These ruptures, which are situated in the meshes of the capillaries, are brought about by (*a*) undue stretching and weakening of these alveolar bases during overdistention of the part with air, and (*b*) a pressure gradient from alveolus to vascular sheath. The gradient is a direct result of the overdistention, which has the effect of pulling away the enveloping alveoli from the pulmonic blood vessels and so of diminishing the pressure in the sheaths around them.[19c]

He found no examples of grossly torn or ruptured alveoli and concluded that the alveolar ruptures are probably small, numerous and widespread. He stated further that after entering the perivascular sheaths the air travels in these sheaths along the pulmonic arteries and veins to the mediastinum. In so doing it compresses both the arteries and the veins. On reaching the mediastinum the air spreads to the mediastinal pleura and then to the subcutaneous tissues of the neck, the retroperitoneal tissues and the pleura. By using colored fluids, he showed that air entered the left pleural cavity through its posterior mediastinal wall in 1 case and the right pleural cavity through the mediastinal wall in another. He stated that these minute ruptures of the blebs of the mediastinal pleura are difficult to locate unless special precautions are taken. The formation of subpleural blebs which extend from the sheaths along connective tissue trabeculae, and which may rupture and produce pneumothorax, although described by some authors, was not seen by him. Furthermore, by inducing artificial tension

19. Macklin, C. C.: (*a*) Pneumothorax with Massive Collapse from Experimental Local Over-Inflation of Lung Substance, Canad. M. A. J. **36**:414, 1937; (*b*) The Site of Air Leakage from the Lung Alveoli into the Interstitial Tissue During Local Over-Inflation in the Cat, Anat. Anz. **85**:78, 1938; (*c*) Transport of Air Along Sheaths of Pulmonic Blood Vessels from Alveoli to Mediastinum: Clinical Implication, Arch. Int. Med. **64**:913 (Nov.) 1939.

pneumothorax in a cat, he was unable to produce mediastinal emphysema. He concluded that the pneumothorax is consequent on the bursting of the overstrained mediastinal wall. The extension from the mediastinum to the subcutaneous tissues and the formation of pneumothorax are salutary in diminishing pressure on the blood vessels in the mediastinum. The pneumothorax closes the alveolar leaks.

Wiethe [7] reported 2 cases of pneumothorax in a series of 50 tracheotomies. During exposure of the trachea in 1 of these cases there were sounds suggesting aspiration of air into a vein. In each instance there was complete pneumothorax on the left side followed by recovery. In his study on cadavers of children he determined that under normal conditions the pleura could be injured only with difficulty during tracheotomy. However, roentgenologic studies made in cases of expiratory obstruction showed that the pleural apexes rose considerably above the clavicles. By injecting a solution of formaldehyde into the trachea under pressure, he showed that the apex of the pleura could easily be injured in performing a low tracheotomy.

Boyd [20] observed air escaping into the interstitial tissues of the lung, collecting here in the form of tiny beads and then passing into the mediastinum.

Schoner and Ehrlich [21] expressed the opinion that during normal respiration the gliding of the visceral over the parietal pleura causes continuous shedding and regeneration of the pleural mesothelium. A slight defect in regeneration accompanied by increased respiratory effort may cause a tear in the visceral pleura and thus produce pneumothorax.

Bullowa,[22] describing the occurrence of subcutaneous emphysema without pneumothorax during an epidemic of influenza, suggested that rupture of the alveoli in those parts of the lung which were free from pneumonia and were overdistended by inspired air was responsible for the subcutaneous emphysema.

Torrey and Grosh,[23] working in an army camp during the epidemic of influenza, made the following observation:

> . . . generalized pulmonary emphysema . . . intense dyspnea . . . and erection of the chest in a state of hyperinspiration, with only tidal air movement. [There was] marked stasis of all the veins entering the chest . . . Cyanosis became more profound. . . . When in apparent respiratory extremis, frequently a patient would begin to complain of pains, substernal and in the jugular fossae,

20. Boyd, W.: A Textbook of Pathology, ed. 3, Philadelphia, Lea & Febiger, 1938.

21. Schoner, A., and Ehrlich, D. E.: Spontaneous Pneumothorax, M. Bull. Vet. Admin. **11**:206 (Jan.) 1935.

22. Bullowa, J. G. M.: Tissue Emphysema in Influenza, M. Rec. **95**:346, 1919.

23. Torrey, R. G., and Grosh, L. C.: Acute Pulmonary Emphysema Observed During the Epidemic of Influenzal Pneumonia at Camp Hancock, Georgia, Am. J. M. Sc. **157**:170 (Feb.) 1919.

and crepitation would be noted in the subcutaneous tissue at the root of the neck, and immediate marked subjective relief was apparent, rapidly followed by a noticeable improvement in the respiratory excursion of the chest, and the most striking decrease in cyanosis and jugular distention. . . . Cutaneous emphysema soon became diffuse, not infrequently spreading down to the pubes. . . . As the intrathoracic pressure was thus relieved these apparently moribund patients often went on to recovery. . . . A number of the patients dying in this stage of early emphysema came to autopsy. The cause of death was evidently the mechanical obstruction of the return flow of the venous blood. [The thoracic contents showed] . . . large emphysematous bullae of the lung and particularly of the mediastinal tissue. . . . The emphysema was such that the lungs could not be collapsed by pressure.

Berkley and Coffen [24] observed cases of extensive subcutaneous and pulmonary interstitial emphysema during the influenza epidemic. From their autopsy material they described two possible routes for the passage of air from the lungs to the chest wall, as follows:

1. The Intrapleural Route: [Air] must pass through both the visceral and the parietal pleura. To pass through . . . without causing pneumothorax it must be assumed that adhesions between these membranes exist at some area and that the passage of air occurs at this point. At necropsy this seemed the possible route in but one case. In this case air could easily be forced out of the lung at a point where recent adhesions had formed and where the parietal pleura at the corresponding point was found to be lifted up by air. However, . . . this is rarely the route taken.

2. Extrapleural Route: For the air to gain access to the mediastinum from the lung without passing through the pleura, it would be necessary to assume a rupture of an air sac and a dissection from this sac back to the hilum. Ruptured air sacs in the peripheral borders of the lung have been found frequently at necropsy. In many cases numerous adjacent air sacs have ruptured, forming a large air space, or "emphysematous excavation." . . . These excavated areas were found to be . . . covered by tense pleura. Leading off from these excavations were "air streaks" . . . following the course of blood vessels. Air could readily be forced from the air spaces to the air streaks. . . . These streaks could be traced along the course of blood vessels to the hilum. . . . By squeezing the lung [under water] . . . bubbles of air arising from around blood vessels [were seen to be forced out at the hilum.] . . . The air may then travel up to the neck. . . . [In 2 cases the pleura covering the tense air space ruptured and caused pneumothorax.]

Jessup [25] stated that in a case of tension pneumothorax air can reach the mediastinum by breaking through the pleural reflection at the hilus, contrary to the observations of Macklin in his animal experiments, or through a laceration in the parietal pleura and then along the endothoracic fascia to the mediastinum.

Iglauer [5] described the case of a child with severe inspiratory distress. Before opening the trachea he noted bubbles of air at the lower angle

24. Berkley, H. K., and Coffen, T. H.: Generalized Interstitial Emphysema and Spontaneous Pneumothorax, J. A. M. A. **72**:525 (Feb. 22) 1919.

25. Jessup, P. M.: Mediastinal Emphysema, Arch. Surg. **23**:760 (Nov.) 1931.

of the wound. The air seemed to be drawn into the wound and then bubbled out. Immediately after tracheotomy the child collapsed, became almost pulseless and had extreme pallor. One hour after operation roentgenologic examination showed right-sided pneumothorax, with shift of the mediastinum to the left. There was slight subcutaneous emphysema of the neck. He cited Champneys [1] in explanation of what had occurred.

Neffson and Wishik [8] cited 4 cases of pneumothorax complicating tracheotomy and 2 cases of pneumothorax accompanying acute laryngeal obstruction due to diphtheria in which no tracheotomy was performed. They explained: [8b]

> . . . the air was sucked into the wound and passed alongside the trachea into the mediastinum, later rupturing through the pleura to cause the pneumothorax. All these patients had subcutaneous and mediastinal emphysema. . . . The occurrence of such emphysema following tracheotomy is due to the fact that the obstruction . . . was only partially relieved by the operation. As a result, the retractions continued and led to the sucking in of air through the wound.

In discussing the 2 cases of pneumothorax and several cases of mediastinal and subcutaneous emphysema (without tracheotomy) occurring in their series of cases of diphtheritic croup, they stated that severe expiratory effort against obstruction leads to alveolar distention and rupture.

> Air, extravasated into the cellular tissue of the lung, spreads along the bronchi and vessels to the hilus and thence to the mediastinum. . . . [The air then passes up to the neck], where crepitations can be felt. It may then spread in all directions. . . . [In one case it extended] from the scalp down to the wrists and groins. [It can pass from the mediastinum to the retroperitoneal tissues.] Just as air extravasates into the cellular tissues of the lungs, it may appear immediately subjacent to the visceral pleura. Rupture of such a vesicle leads to pneumothorax, which occurred in two cases.[8a]

Neffson and Bullowa,[10] describing a case of bilateral tension pneumothorax associated with influenza, commented on the mechanism of production of the pneumothorax and extensive subcutaneous emphysema as follows:

> The obstruction of the bronchioles by exudate and the marked supraglottic edema combined to produce violent inspiratory efforts. The dilatation of the bronchioles caused by the strenuous inspiratory efforts and the concomitant diminution of their contractile . . . power caused by the influenzal infection resulted in overdistention and coalescence of the functioning pulmonary alveoli. . . . As a result, . . . certain of the emphysematous lobules burst. If they were subpleural, air passed into the pleura. . . . If the tear is not into the pleura, the air is forced into the pulmonary interstitial tissue and travels along the loose tissue surrounding the blood and lymph vessels and bronchi to the root of the lung, where it either ascends between the mediastinal fascial planes and appears in the neck, spreading down from this point or, more rarely, descends into the retroperitoneal space and perirenal tissues. If there are adhesions between the visceral and the parietal pleura, air may appear in the thoracic wall without traveling down from the neck.

Dolgopol and Stern [12] described the case of a 2½ year old boy who had undergone tracheotomy for foreign body in the larynx at the age of 1 year and who just before admission to the Willard Parker Hospital was again tracheotomized, this time because of laryngeal diphtheria. He was moribund on admission, with extensive faucial and laryngotracheobronchial diphtheria. Two days after tracheotomy routine roentgenograms showed no pneumothorax or pneumonia. During this period large pieces of membrane were aspirated from the trachea and bronchi. On the third day there appeared subcutaneous emphysema of the left side of the neck and chest and signs of pneumothorax, on the left side, confirmed roentgenographically. Air was withdrawn from the left pleural cavity on several occasions, but the child died. The significant postmortem observations were as follows:

Because of previous empyema, the left lung was partially adherent, so that it did not completely collapse. Microscopic examination showed alveolar emphysema, interstitial emphysema, with large air bubbles present in the widened, edematous interlobular septums, and air in the perilymphatic spaces. There was no air in the perivascular sheaths, such as Macklin noted. No mediastinal emphysema was present. The authors concluded that since there was no mediastinal emphysema in this case, but air was found in the perilymphatic spaces of the interlobular septums, it was probable that the air traveled from the septal lymphatics, not to the hilus (as explained by Macklin [10] in cases of perivascular transportation) but to the periphery of the lung, and then escaped through the pleural adhesions into the pleural cavity and into the retropleural connective tissue. The air could also have found its way into the pleura through rupture of the retropleural blebs. From the retropleural tissues the air could readily have spread into the subcutaneous tissues of the neck and into the abdomen.

Michels [11] reported 5 cases of pneumothorax and 1 case of mediastinal emphysema complicating tracheotomy. In 1 case subcutaneous emphysema preceded the tracheotomy, which was followed by pneumothorax on the right side. In another case tracheotomy was performed after bronchoscopic examination was made for a foreign body. Pneumothorax occurred on the right side. In a third case pleura protruded into the wound and a hissing sound due to rupture was heard. The trachea was then incised. Later subcutaneous and mediastinal emphysema developed, but not pneumothorax. In all 3 cases recovery occurred. There were 3 cases of bilateral pneumothorax, with death occurring in all. Postmortem examinations revealed the following changes:

Case 1. Death one day after tracheotomy. Pneumothorax on the right side; emphysema of the lower lobe and atelectasis of the upper lobe of both lungs; large quantity of air in the posterior mediastinum, with large air blebs extending upward to surround the trachea. Stenosis of the larynx.

Case 2. Death five hours after tracheotomy. Bilateral pneumothorax; severe emphysema of the mediastinum; pulmonary atelectasis. Necrotic membrane over the larynx, trachea and bronchi.

Case 3. Death two days after tracheotomy. Pneumothorax on the left side; massive air blebs along the large pulmonary vessels; numerous blebs in the anterior pericardium; no air around the trachea or larynx. (The child struggled during the operation. Pleura protruded into the wound. A definite hissing sound of pleural rupture was heard.)

Michels concluded that the occurrence of pneumothorax and mediastinal emphysema with increased intrapulmonic pressure can best be explained as follows:

> The extrapleural route, with dissection of minute globules of air from the interstitial tissues along the vessels to the hilus, is a more comman cause of their occurrence with increased intrapulmonic pressure. . . . The dissection of air by way of the pretracheal fascia to the mediastinum, aided by the negative inspiratory pressure, affords another avenue of entry. The possibility of direct injury to the pleural dome during tracheotomy cannot be ignored.[11]

Graebner,[14] reporting 3 cases of pneumomediastinum and 2 cases of pneumopericardium associated with acute obstructive laryngitis, stated that the escape of air into the mediastinum takes place by the route suggested by Macklin. In 1 case tracheotomy was not performed; in another bubbles of air were seen to escape on separation of the ribbon muscles over the trachea, so that ". . . there was already present a positive air pressure in the mediastinum." In the other 3 cases the mediastinal emphysema occurred from twenty-four to thirty-six hours after tracheotomy, when bronchopneumonia and abundant secretions caused respiratory embarrassment. He concluded that the occurrence of mediastinal emphysema is secondary to the obstruction in the respiratory tract and is not the result of tracheotomy per se.

> [The air was] passed out of the substance of the lung and along the perivascular sheaths into the mediastinum by excess positive pressure incident to respiratory obstruction [rather than being] sucked in through the tracheotomy wound by inspiratory efforts and normal negative pressure in the mediastinum.

MECHANISMS IN PRODUCTION OF TENSION PNEUMOTHORAX, MEDIASTINAL EMPHYSEMA AND SUBCUTANEOUS EMPHYSEMA FOLLOWING TRACHEOTOMY

From the experimental, clinical and pathologic material presented, the production of tension pneumothorax and mediastinal and subcutaneous emphysema can be explained as occurring by the following routes:

1. Intrinsic Route (this can occur also without tracheotomy): Increased intrapulmonic pressure caused by trapping of air results in distention and, finally, in rupture of the pulmonary alveoli. This was noted in nontracheotomized patients by Berkley and Coffen,[24] Torrey and Grosh[23] and Kelman[17] during the epidemic of influenza, and in

tracheotomized patients by Neffson and Wishik [8]; described in post-mortem reports (cases 3 and 5) in this series and by Dolgopol and Stern,[12] and noted in animal experiments by Kelman,[17] Joannides and Tsoulos [18] and Macklin.[19]

Such ruptures may be microscopic, numerous and widespread (Macklin [19b]) or gross and limited to one area (Berkley and Coffen [24]). From these ruptures the air can then spread into the following tissues:

A. Pulmonary interstitial tissue, around the capillary meshwork (Macklin [19c]). Thence it can proceed in two directions.

1. To the hilus of the lung, in the perivascular and perilymphatic spaces of the interlobular septums (Macklin; [19a] Boyd,[20] Dolgopol and Stern [12]), along the pulmonary vessels to the mediastinum, producing mediastinal and pericardial (Graebner [14]) emphysema. From the mediastinum the air can proceed in three directions.

(*a*) Laterally. Rupture of mediastinal blebs into the pleural cavity result in pneumothorax (Joannides and Tsoulos,[18] Macklin [19a]). Another possibility is the passage of mediastinal air through small dehiscences in the parietal pleura, particularly where the visceral and the parietal pleura join, resulting in pneumothorax (my theory).

(*b*) Upward, along the deep cervical fascial layers around the trachea and larynx, breaking through into the subcutaneous tissues of the neck.

(*c*) Downward, through the diaphragmatic openings to the retroperitoneal tissues of the abdomen (Neffson and Wishink;[8] Macklin [19a]).

2. To the periphery of the lung, in the perilymphatic spaces of the interlobular septums, through pleural adhesions into the pleural cavity or into the retropleural connective tissue. Rupture of retropleural blebs into the pleural cavity can produce pneumothorax. From the retropleural tissues air can ascend to the subcutaneous tissues of the neck or descend to the retroperitoneal tissues (Dolgopol and Stern; [12] [in part] Berkley and Coffen [24]).

B. Subpleural tissue, forming subpleural blebs which burst (Berkley and Coffen [24]; Torrey and Grosh [23]; Neffson and Wishik [8]); case 3 in this series, noted post mortem, and produce pneumothorax. This is uncommon, however. Rupture through a pleural adhesion can cause retropleural, and subsequently subcutaneous, emphysema without producing pneumothorax.

II. Extrinsic Route: Owing to inspiratory obstruction, the negative pressure in the thorax is increased, and air is sucked through the tracheotomy wound along the deep cervical fascia into the mediastinum (noted by Champneys [1] and confirmed by Iglauer,[5] Neffson and Wishik,[8] the Jacksons [8] and Michels [11]). From the mediastinum the air can reach the pleura, neck and abdomen in the same manner as described for the intrinsic route. This route is probably the more common after tracheotomy.

Since pneumothorax occurs so often shortly after tracheotomy—in 60 per cent of cases it was noted within a few hours after operation—it would be hard to deny a consequential relationship. Of course, it can be argued that increased respiratory efforts during tracheotomy may lead to increased intrapulmonic tension, which is further augmented by the sudden inrush of air following the insertion of the tracheotomy tube, so that rupture of pulmonary alveoli occurs, with spread by the intrinsic route. However, that this is the less likely manner of spread is shown, first, by the rarity of pneumothorax following intubation, during which there are also increased respiratory effort and a sudden inrush of air, though to a lesser degree, and, second, by the postmortem material in this series and in that of Michels,[11] in which pulmonary interstitial emphysema resulted in only 2 out of the 9 cases, mediastinal emphysema alone occurring in the rest.

III. Traumatic Route: According to Wiethe,[7] injury to the dome of the pleura during tracheotomy, when there is pronounced expiratory obstruction, is a strong possibility. However, actually it is probably rare. A hissing sound, as of air being sucked in, is rarely described, and when actually heard has not always been followed by pneumothorax (cases of Michels[11]). Furthermore, should this occur, it would not be likely to lead to tension pneumothorax; that is, the air pressure in the pleura would not be greater than atmospheric, since the air could escape through the pleural tear. Also, I have found no evidence of such an accident in any autopsy. Moreover, the pleura is higher on the right than on the left; yet in my series the pneumothorax was on the left in 5 of the cases and on the right in only 3. Last, if trauma were an important factor, there would be little expectation of lowering the incidence of pneumothorax from 25 to 8 per cent, as occurred in this series.

MANAGEMENT

Prophylaxis.—Since the most frequent cause of pneumothorax and mediastinal emphysema following tracheotomy appears to be the sucking in of air through the tracheotomy wound into the mediastinum, the prevention of this occurrence is the prime consideration. This can be accomplished by (*a*) eliminating respiratory obstruction during operation, (*b*) eliminating postoperative respiratory obstruction and (*c*) avoiding the opening of avenues for the tracking down of air into the mediastinum.

The elimination of respiratory obstruction during tracheotomy can be accomplished by passing a metal tube into the trachea before the operation, thus providing an adequate airway and converting a gasping, restless, struggling child into a quiet one, permitting an unhurried operation.

After operation, the airway must be kept clear religiously at all times. In several cases pneumothorax has developed from two to four days

after tracheotomy, when the airway was not or could not be kept open continuously, in spite of repeated aspiration.

The chances of air tracking down into the mediastinum can be reduced by the following precautions: (*a*) Avoiding an anatomic dissection, with demonstration and wide spreading of all the tissues, particularly the pretracheal fascia. Instead of separating the pretracheal fascia, this fascia and the tracheal rings should be incised at the same time. This should be done from below upward in order to avoid the possibility of nicking the pleura. (*b*) Application of iodoform packing firmly around the tracheotomy wound to seal off the points of entry of air.

Jackson stated that suturing of the skin tends to trap the air and encourages the formation of mediastinal emphysema. From my experience this observation cannot be confirmed.

Intubation was not a factor in causing an increase in pneumothorax—as a matter of fact, there were proportionately one and one-half times as many cases of pneumothorax among nonintubated patients as among intubated ones.

Diagnosis.—Early diagnosis is essential. Failure to recognize pneumothorax is the cause of needless deaths. The diagnosis depends on the clinical picture, the physical signs and roentgenographic evidence. The clinical picture will depend on the rapidity of onset and the magnitude of the pneumothorax and on whether it is unilateral or bilateral. Thus, patients may be almost symptom free, or they may go into shock, become deeply cyanotic and stop breathing.

The physical signs are: (1) dyspnea, with diminution in the volume and force of the blast from the tracheotomy tube, the examiner making certain that the airway is clear; (2) diminished excursion of the affected side, with displacement of the heart to the opposite side, and (3) diminished breath and voice sounds, with hyperresonance or tympany.

Pneumothorax may be mistaken for pulmonary emphysema, and therefore in all cases roentgenograms should be made routinely as soon after tracheotomy as is feasible and as often thereafter as seems warranted.

During tracheotomy the presence of a pleural bubble in the wound, enlarged with each expiration, can be taken as good tentative evidence of the presence either of pneumothorax or of mediastinal emphysema, and immediate roentgenograms should be taken for confirmation. Two patients, who later were found to have pneumothorax, stopped breathing before the tracheotomy; 1 became cyanotic and went into shock immediately after tracheotomy, and 1 died on the operating table. Accidents of this type should, therefore, be a warning to the operator to look for pneumothorax as the cause.

Mediastinal emphysema can be recognized by the crepitation that is heard with each heart beat if the stethoscope is held firmly pressed to the cardiac area. The apex beat may be lost to inspection and palpation.

With severe emphysema there are extreme cyanosis and dyspnea, rapid pulse, congestion of the superficial veins of the neck, a low blood pressure, substernal discomfort and pain. The roentgenograms reveal fuzziness and streaking in the mediastinum and around the cardiac borders.

Treatment.—In the case of unilateral pneumothorax of slight extent, or even of bilateral pneumothorax which causes no embarrassment, nothing is necessary except careful observation. The air in the pleural cavity is usually absorbed within a few days and does not affect the outcome. When tension pneumothorax is present, that is, when air can get in but cannot get out, and the pressure is above atmospheric, immediate and continuous or repeated decompression is necessary. Failure to do this results in mass venous stasis, due to increased intrathoracic pressure, and anoxia. Air embolism may also occur as a complication.[18] The decompression should be done slowly, with a minimum of manipulation in order to avoid a too rapid shift in mediastinal pressure, the possibility of air embolism, injury to the lung and pleural shock. One of the best methods is by means of closed, underwater drainage. A large, dull needle, a blunt cannula or a narrow rubber catheter is inserted into the affected pleural cavity and is connected with rubber tubing, the distal end of which is placed in an antiseptic solution, such as a 1 : 10,000 solution of potassium permanganate, about 18 inches (45 cm.) below the chest. If a piece of glass tubing is interposed in the rubber tubing, the fluid will be seen to rise and fluctuate freely when the free air has been removed. At this point, the tubing may be clamped and the patient watched. If there is no distress over a period of time, the needle can be withdrawn from the chest. If, for some reason, this procedure presents difficulties, a finger cot, with the tip cut off, can be attached to the hub of the needle. This will act as a one way, flapper valve, allowing the air to get out, but not to come in. A third method is to use a pneumothorax apparatus and remove enough air to bring the pressure down to the normal of — 6 to — 10 cm. of water and to repeat this as often as necessary. If needed, oxygen can be given through the tracheotomy tube by means of a small catheter or funnel placed over the opening.

In the case of mediastinal emphysema, Tiegel,[26] and later Thorek,[27] advised making an incision in the anterior aspect of the infrathyroid region down to the trachea and using a suction apparatus to withdraw air from the mediastinum. Since the incision is already there, a rubber catheter can be inserted into the mediastinum if necessary. However, I have never had to resort to this.

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26. Tiegel, M.: Ein einfaches Verfahren zur Bekämpfung des Mediastinalemphysems, Zentralbl. f. Chir. **38**:420, 1911.

27. Thorek, M.: Modern Surgical Technic, Philadelphia, J. B. Lippincott Company, 1938, vol. 2.

PROGNOSIS

Unilateral pneumothorax carries with it a relatively low mortality rate, less than 20 per cent (cases in this series; Michels,[11] and others), whereas bilateral pneumothorax has a very high mortality, over 90 per cent (cases in this series; Michels,[11] and others). Early recognition, together with immediate and continuous removal of the compressed air, will result in the saving of many lives.

SUMMARY AND CONCLUSIONS

In a review of 126 consecutive cases of tracheotomy performed at the Willard Parker Hospital for the relief of acute nondiphtheritic obstructive infections of the larynx, trachea and bronchi, 17 cases of pneumothorax, an incidence of 13.5 per cent, were found. These cases are analyzed as to age, sex, site (right, left, bilateral), time of occurrence after tracheotomy, relationship to previous intubation, incidence in relation to frequency of tracheotomy during various periods, time required for the disappearance of free air, mortality rates under various conditions and postmortem observations.

(*a*) With an increase in the relative frequency of tracheotomy from 4.3 per cent, in the first five years, to 19.4 per cent, in the last three years, there was a concomitant lowering of the incidence of pneumothorax from 25 to 8 per cent.

(*b*) Pneumothorax occurred one and one-half times as often in cases in which intubation was not done prior to tracheotomy as in cases in which it was done.

(*c*) The mortality rate of unilateral pneumothorax is less than 20 per cent; that of bilateral pneumothorax, over 90 per cent.

Experimental work, clinical and pathologic material and discussions found in the literature are reviewed, and a conception of the mechanisms involved in the production of tension pneumothorax, mediastinal emphysema and subcutaneous emphysema is presented.

Early recognition and immediate treatment of this condition are life saving. The possibility of the occurrence of tension pneumothorax must always be borne in mind and watched for after tracheotomy. Warning signs of pneumothorax and mediastinal emphysema are described, and the prophylaxis and treatment are outlined.

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DIAGNOSIS AND TREATMENT OF MÉNIÈRE'S SYNDROME

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Any new method of treatment for Ménière's syndrome is apt to be regarded askance, not without reason in view of the failure of most of the methods suggested so far to justify the high hopes which have been set on them at birth. The observations which follow are offered with the realization that, though the principle on which they are based has survived for two years and continues to prove satisfactory in practice, it is not therefore of necessity the whole answer to the problem. It is believed, however, that it may be at least part of it.

There are two outstanding reasons for the failure of any particular treatment to achieve its hoped-for results. One lies in laxity of diagnosis. Instead of keeping strictly to the strait and narrow path and demanding that only those cases which present the classic triad of symptoms of recurring vertigo, deafness and tinnitus shall be acceptable as instances of Ménière's syndrome, too many diagnosticians allow themselves to be lured into loose thinking by some such facile cloak for ignorance as pseudo Ménière's disease. Under this elastic heading are placed cases of paroxysmal vertigo which is not accompanied by deafness and tinnitus and for which no satisfactory explanation can be found. Instead of an honest designation "vertigo of unknown origin" the current euphemism is pseudo Ménière. This term may include such diverse disease processes as hyperpiesis and multiple sclerosis. Yet by its use of the name Ménière it links in the mind the false condition with the genuine article and so produces confusion of thought, carelessness in diagnosis and failure in treatment. It is a great pity that the term was ever coined. If any order is to come out of chaos, the first thing is to do away with the term pseudo Ménière, or pseudo anything else, for that matter. "Pseudo," the false or spurious, has no more place in medical terminology than it has in scientific inquiry.

The other reason for failure of treatment is the tendency, partly unconscious, to regard the malady of Ménière as a disease *sui generis*. Lip service is paid to a syndrome, but thought centers on a disease and the treatment for it, usually the last one publicized. It must be empha-

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sized that the triad of symptoms commonly known as Ménière's can be produced by more than one etiologic factor, and that the first requisite in the investigation of any case is to classify it correctly. To attach the label Ménière's syndrome is not the end but the beginning of diagnosis.

In this communication, Ménière's syndrome and Ménière's syndrome only will be discussed—Ménière's syndrome according to the strictest letter of the law. It is hoped to show that cases of the syndrome can be divided into certain groups according to causation and that each group demands its appropriate treatment.

DIAGNOSIS

That accuracy of diagnosis is essential to efficient treatment is a truism which is sometimes disregarded because, though worthy of all acceptation, it is yet at times a hard saying. This is particularly the case in the diagnosis of Ménière's syndrome. The history is so characteristic, and the results of physical examination apart from the deafness so frequently negative, that the temptation is to make the diagnosis on history and deafness alone. It is a dangerous practice. Only by the most thorough examination and a process of exclusion can accurate diagnosis be achieved.

1. *Neurologic Lesions.*—In the first place, every case of Ménière's syndrome demands a careful neurologic examination. Moreover, in cases in which this examination gives negative results and yet treatment along the lines to be described fails to relieve, neurologic examination must be repeated at regular intervals. Signs may appear as time goes on. Paroxysmal vertigo with all the attributes of Ménière's syndrome can occasionally be the first, and for some time the only, symptom of a lesion in the cerebellopontile angle. I have encountered 3 such cases in the past two years. This situation is a trap into which it is all too easy to fall. The moral is never to make a diagnosis on the history alone.

2. *Bárány Tests and Stricture of Eustachian Tubes.*—An equally careful otologic examination is the next step. This examination may well omit Bárány tests except when there is evidence of an intracranial lesion. In the common run of cases, information of value will be obtained by them only in the presence of neurologic complications, and then only if testing is done by an accurate quantitative method, such as the one I have described.[1] Admittedly, a careful quantitative test will sometimes give helpful information in difficult cases,[2] but

1. Atkinson, M.: A Simple Quantitative Method of Testing Vestibular Function, Arch. Otolaryng. **30**:916 (Dec.) 1939.

2. Atkinson, M.: Changes in Vestibular Sensitivity in Ménière's Syndrome and Their Significance, Arch. Otolaryng. **33**:969 (June) 1941.

it is waste of time to do Bárány tests in every case, to demand them as a routine in all cases of dizziness.

The otologic examination must, however, include a careful appraisal of relative patency of the eustachian tubes. This is admittedly a reversal of the generally accepted roles, in which Bárány tests are considered *de rigueur* and inflation of the eustachian tubes an outmoded procedure. But, as has been said, Bárány tests seldom help in diagnosis, whereas stricture of the eustachian tube is the direct cause in an appreciable number of cases. From time to time complete obstruction from swelling of the mucosal lining may take place and so produce an acute alteration in pressure in the middle ear and labyrinth. The result is a labyrinth storm with deafness and tinnitus—a Ménière attack. Recurrence of such an attack can be prevented entirely by gradual dilation of the stricture, in the same way that a urethral stricture is dilated, with a bougie. In many cases no other treatment is necessary, despite the skeptics.[3]

3. *General Causes.*—The third step is a thorough general examination to determine the presence of any associated condition, in particular of any focus of infection, especially in the nose and throat.

Of associated conditions apart from infections, the two to be particularly borne in mind are alterations in blood pressure and disease of the gallbladder. With regard to the former, a low blood pressure is as frequent a cause of vertigo as a high one, though neither is common. Each or either demands attention to the cause. The latter condition is not infrequently associated with Ménière's syndrome, and its remedy, whether by medical or by surgical means, will at times have an appreciable effect on the attacks of vertigo. However, resort is not to be had to the knife in the first place unless the necessity for it is beyond a doubt, for the treatment appropriate to the Ménière attacks will in many instances relieve the gallbladder symptoms. The two conditions are so closely connected, by what exact metabolic mechanism is not yet known, that the exact diagnosis is sometimes in doubt and not infrequently is made incorrectly, usually disease of the gallbladder in mistake for Ménière's syndrome.

As to focal infection producing a so-called toxic labyrinthitis, my own opinion is that its incidence and its effect have been greatly exaggerated in Ménière's syndrome as in many other conditions in which it is regarded as a causative factor. That a nasal sinusitis may on occasion be the cause of a tubal catarrh which will produce a block of the eustachian tube and so vertigo I do not deny; that it does it at all frequently I know is not so. In this series no case has been met with in which there was a focus of infection which needed removal. However,

3. (*a*) Atkinson, M.: Vertigo, Canad. M. A. J. **42**:326 (April) 1940. (*b*) Woodman, E. M.: The Position of the Portmann Operation in Relation to Labyrinthine Vertigo, Proc. Roy. Soc. Med. **32**:1642 (Oct.) 1939.

Wright [4] maintained that all cases of Ménière's syndrome own this etiologic basis, and though this is undoubtedly an extreme view, it is certainly wise to be rid of gross foci of infection, if only from the point of view of general health. This does not justify the routine removal of questionably infected tonsils, teeth or other organs in the dubious hope of ultimate benefit.

4. *Idiopathic Group.*—There is left, after elimination by these three steps, that large group of cases in which the disorder is the one commonly thought of as Ménière's disease and may be called idiopathic. It is in regard to this group in particular that thought has tended to run along a single line, looking for one cause and one treatment,[5] instead of continuing in terms of a syndrome and looking for two or more. In a previous paper, I have put forward evidence to show that this large group of cases can be divided into at least two divisions, each owning a different cause,[6] and it is on this group that I wish from now on to focus attention.

(*a*) Some cases of Ménière's syndrome own an allergic basis. The concept of allergy as the cause of Ménière attacks was put forward originally, I believe, by Dohlman,[7] and has proved partly true. It does not, however, explain all, or even the majority of, cases, and the third step in the investigation of a case of Ménière's syndrome is to eliminate allergy as a cause. In the search for a simpler means of doing this than by multiple cutaneous tests, which, especially in cases of general allergy, are notoriously unreliable, or by trial and error in the case of food allergies, which may result in imminent starvation without other definite result, the idea was hit on to test for sensitivity to histamine by the cutaneous method, on the theory that histamine is a type substance in production of allergic manifestations (table 1). The method proved satisfactory in that it separated a group of cases in which the patients were sensitive to histamine from another group in which they were insensitive, and its validity has been demonstrated in practice by the reactions to treatment.[7a]

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4. Wright, A. J.: Further Clinical Observations on the Nature and Treatment of Ménière's Disease, Proc. Roy. Soc. Med. **33**:459 (June) 1940.

5. Furstenberg, A. C.; Lashmet, F. H., and Lathrop, F.: Ménière's Symptom Complex: Medical Treatment, Ann. Otol., Rhin. & Laryng. **43**:1035 (Dec.) 1934. Mygind, S. H., and Dederding, D.: Les syndromes ménièriques, Paris, Presses universitaires de France, 1934. Talbott, J. H., and Brown, M. R.: Ménière's Syndrome: Acid-Base Constituents of the Blood; Treatment with Potassium Chloride, J. A. M. A. **114**:125 (Jan. 13) 1940.

6. Atkinson, M.: Observations on the Etiology and Treatment of Ménière's Syndrome, J. A. M. A. **116**:1753 (April 19) 1941.

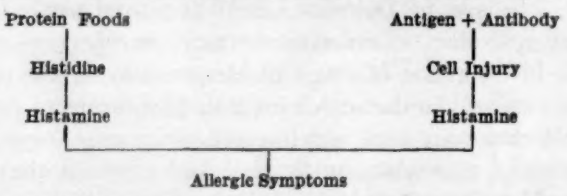
7. Dohlman, G.: Menieresche Krankheit auf allergischer Basis, Acta otolaryng. **27**:245, 1939; Studies of a Vestibular Syndrome Induced in Guinea Pigs by Allergic Reaction, ibid., 1939, supp. 32.

(Footnotes continued on next page)

The method is simple. An intradermal test is performed by injection of 0.0057 mg. of histamine base (0.01 mg. of histamine dihydrochloride). In the normal person a wheal, white in the center, appears immediately, which in five minutes has changed to a yellowish wheal ⅓ to ½ inch (0.8 to 1.3 cm.) across, with definite edges and surrounded by a flare of erythema 1¼ to 1½ inches (3 to 3.8 cm.) in diameter. The wheal begins to fade in ten minutes, is faint in fifteen minutes and has disappeared in twenty minutes. Such a result is negative; the patient is insensitive to histamine (fig. 1 *a*).

In the sensitive person the wheal attains a larger size, ½ to ¾ inch (1 to 1.9 cm.), with a surrounding flare of 2 to 2½ inches (5 to 6.4 cm.). and, in addition, one or more long trailing pseudopods appear, 1 inch or more in length (fig. 1 *d*). The pseudopod is definite at the end of five minutes and does not start to fade until the end of fifteen or twenty

TABLE 1.—*Theoretic Conception of Allergy*



minutes, often being still apparent as a red streak at the end of thirty minutes. Such a result is positive; the patient is sensitive to histamine.

There are intermediate appearances. Figure 1 *b* shows three small buds disturbing the regularity of the edge of the wheal. These are not to be mistaken for small pseudopods—such an appearance does not indicate even a minor degree of sensitivity; it is to be read as definitely negative. Figure 1 *c* is more difficult to interpret. Mostly, such an appearance indicates a negative reaction in a person more than usually susceptible to vasodilator drugs. But it may indicate sensitivity in minor degree, and it is therefore advisable to repeat the test in forty-eight hours (not less because of a negative phase) with double the dose of histamine.

7a. This would seem to imply that the histamine cutaneous test is a reliable indication of the presence or absence of sensitivity in a given subject. Though at first this was believed to be true, it is now known not to be so. Persons with specific sensitivity as definite as true hay fever may and often do give negative reactions to this test. Nevertheless, the results of the test are valid in that they divide patients with Ménière's syndrome into two groups, the one sensitive and the other insensitive to histamine. The former can be treated with satisfactory results by desensitization to histamine, while the latter cannot. The precise

The result then is unequivocal; either a definite pseudopod develops and the patient is sensitive, or the same picture appears and the patient is insensitive.

By this means a group of cases in which the patients were sensitive to histamine, comprising about one fifth of the entire number (20 out of 108, table 2), can be separated from the others. It must be emphasized, however, that a rigid adherence to the criteria given is essential to success. To place a case in the wrong group by false interpretation of the test means wrong treatment and worse than disappointment, deterioration, as will be seen.

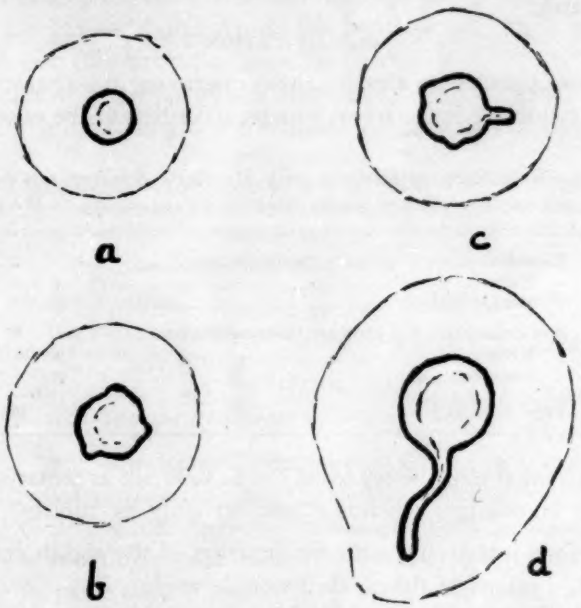


Fig. 1.—Cutaneous reactions to the intradermal injection of 0.0057 mg. of histamine base. In this figure, *a* and *b* indicate negative reactions; *c* shows a doubtful reaction, and *d*, a positive reaction.

(*b*) In the large majority of cases remaining, some four fifths of the entire number (88 out of 108, table 2), the patients were insensitive to histamine, and the question then arises: What in them is the basis of their attacks? If the manifestations of allergy may be regarded, from a purely vascular point of view, as a capillary vasodilator mechanism, and the vertigo in the cases of sensitivity to histamine therefore as of primary vasodilator origin, there are good reasons for inferring that the mechanism in the other group is primarily vasoconstrictor. These

implications of the test, however, remain unknown; their elucidation must wait on a greater understanding of the role played by histamine in the bodily economy in health and in disease.

reasons, and the experimental observations which support them, have been discussed elsewhere.[6] Suffice it to say here that the exhibition of vasoconstrictor drugs has precipitated attacks of vertigo in members of this group and the administration of vasodilator drugs has relieved them. If, moreover, it is assumed—a reasonable assumption—that the primary vasoconstriction is followed by secondary vasodilatation, as, for instance, in Raynaud's disease, an explanation is available both of the clinical aftermath of unsteadiness, which is so characteristic of Ménière attacks in this group, and of the pathologic observation of dilatation of the endolymphatic system in each of the 3 cases described by Hallpike and associates.[8]

CLASSIFICATION

It is now possible to classify cases displaying the characteristics of Ménière's syndrome into various groups according to the etiologic basis.

TABLE 2.—*Proportion of Patients with Ménière's Syndrome Who Were Sensitive to Histamine to Those Who Were Insensitive to the Drug*

Histamine-sensitive (primary vasodilatation)		20
Males	5	
Females	15	
Histamine-insensitive (primary vasoconstriction)		88
Males	46	
Females	42	
Total (1940-1941)		108

This classification must be regarded not as final but as tentative, expressing present knowledge, which is almost certainly incomplete.

A. Lesions interfering with the function of the eighth cranial nerve
   1. Lesions of the cerebellopontile angle
   2. Degenerative vascular disease

B. Lesions interfering with the function of the labyrinth
   1. Alteration in intralabyrinthine pressure
      (*a*) From without—stricture of eustachian tube
      (*b*) From within—increased production of endolymph (Primary vasodilatation; allergy; sensitivity to histamine)
   2. Vascular disease
      (*a*) Angiospasm; primary vasoconstriction
      (*b*) Arteriosclerosis
   3. "Toxic labyrinthitis"

      Focal infection

8. Hallpike, C. S., and Cairns, H.: Observations on the Pathology of Ménière's Syndrome, Proc. Roy. Soc. Med. **31**:1317 (Sept.) 1938. Hallpike, C. S., and Wright, A. J.: On the Histological Changes in the Temporal Bones of a Case of Ménière's Syndrome, ibid. **32**:1647 (Oct.) 1939.

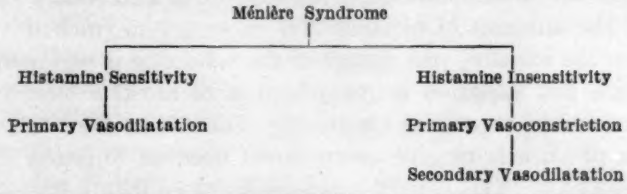
PRINCIPLES OF TREATMENT

The treatment of lesions of the cerebellopontile angle is the concern of neurologists and neurosurgeons. The treatment of stricture of the eustachian tube has already been outlined—gradual dilation of the stricture with a soft bougie. The efficacy of this treatment in suitable cases has been vouched for by others besides myself.[8b] In spite of the doubting Thomases and the disrepute into which eustachian tube bougies have fallen because of their misuse by the inexpert, it can be said without hesitation that in appropriate cases the condition responds admirably.

There remain the cases of the idiopathic type, which is the main subject of discussion. The production of symptoms in this group depends on a dual mechanism (table 3), as has been shown. It is, in fact, two groups, not one (see preceding classification).

(*a*) *Cases of Sensitivity to Histamine* (Primary Vasodilatation).—In cases falling into this group treatment has consisted of desensitization

TABLE 3.—*The Dual Mechanism in Ménière's Syndrome*



to histamine by a slow method described in the paper previously mentioned.[6] The test intradermal dose is repeated subcutaneously, and then at intervals of two to four days the dose is gradually increased as if one were giving a vaccine and with the same consideration for general reactions. The increase must be made slowly and with great care until the tolerance of the patient has been determined, for rather severe reactions can occur. The maximum dose ever used is 0.57 mg. of histamine base (1 mg. of histamine dihydrochloride), though some patients will not tolerate so much and some only much less. Again, individual tolerance must be borne in mind and dosage must not be forced—an overdose may produce a return of symptoms, which can be controlled again by suitable reduction. When the maximum dose attainable is reached, it is repeated at four weekly intervals. A second course is usually necessary after six months or so, and, as far as present knowledge goes, sometimes a third. Experience is not yet sufficient to say how often this is so.

With the rapid method of desensitization described by Alexander and used by Shelden and Horton [9] I have no experience. In view of the

9. Shelden, C. H., and Horton, B. T.: Treatment of Ménière's Disease with Histamine Administered Intravenously, Proc. Staff Meet., Mayo Clin. **15**:17 (Jan. 10) 1940.

reactions which are sometimes produced even by very gradual increase of dose by the slow method, I have hesitated to use it. Further, it demands hospitalization, which is not necessary with the slow method.

(*b*) *Cases of Insensitivity to Histamine* (Primary Vasoconstriction).—Nicotinic acid has been the substance used in treatment in this group, specifically on the grounds of its being a vasodilator, not of its being a vitamin. How far this postulate is true is not yet certain.

That nicotinic acid has a vasodilator action on the peripheral circulation is obvious from the dermal flush it produces. The present evidence, however, is that it has no such effect on the intracranial vessels,[10] of which the auditory artery is one. Moreover, it is beginning to appear as if a patient can become saturated with nicotinic acid. When the dose is gradually increased a point is reached where dermal flush becomes minimal or absent, and if still the dose is increased symptoms may recur and symptoms of overdosage—headaches, intestinal colic and hot flashes—appear, as with histamine in patients who are sensitive to that drug. This suggests that a primary vasoconstrictor mechanism has been corrected and that further stimulation has resulted in a secondary vasodilator action. The amounts of nicotinic acid necessary to reach this point of saturation are variable—the dosage of the substance is highly individual. In practice this variation in the tolerance of nicotinic acid has to be borne in mind in organizing treatment. There is no hard and fast rule. My own practice is to give as an initial dose an injection of 25 mg. intramuscularly. This usually produces no more than a transitory mild flush, lasting five to ten minutes, and even in the unduly sensitive patient does not give rise to an excessive reaction. The dose is then gradually increased to the limit of tolerance, maintained there for a month or more and eventually decreased by slow steps to a maintenance dose, which is continued for many months.

It is impossible to prescribe a definite line of treatment in any particular case because of the individual variation. In general, the line found most useful has been to start, after the test intramuscular dose, with intravenous injections, which may be increased daily to approaching tolerance limit and should be continued for two or three weeks. Then, the patient is taught to give himself intramuscular injections, which he does daily, the dose again being gradually increased to the optimum, while at the same time nicotinic acid is given by mouth. Eventually, and when depends again on the individual patient, injections are diminished in amount and discontinued and oral administration alone is relied on. It is important to proceed slowly, to be patient and persistent rather than to try to rush fences. Too quick an increase, too high a dose, especially if persisted in, is apt to lead to trouble rather than to rapid

10. Myerson, A.; Loman, J., and Rinkel, M.: The Intracranial and Peripheral Vascular Effects of Nicotinic Acid, Am. J. M. Sc. **202**:211 (Aug.) 1941.

relief. Particularly is this the case in the early days of treatment, when sometimes the exhibition of nicotinic acid seems to upset the equilibrium and to make matters worse. Such setbacks are apt to lead to doubt and discouragement. They need not if the physician is certain of the accuracy of his diagnosis, for persistence in treatment and careful adjustment of dose will result in improvement after a short time. In the meantime, the morale of the patient must be bolstered and his confidence maintained, not always too easy a task.

I have said that it is impossible to be dogmatic as to dosage, but a fair average for the maximum dose is 50 mg. by intramuscular injection and 50 to 100 mg. by mouth. Some patients will need more; some, less. The extremes of variation in my own experience have been 10 mg. injected intramuscularly daily, at one end, and, at the other, 400 mg. injected intravenously and 300 mg. given by mouth daily. Both patients at first lost their symptoms, and both eventually overdosed themselves and had a recurrence. Both were physicians—one of 57, who, having the patience of age, adjusted his dose and did well; the other of 26, who, with the impatience of youth, fled to the surgeons. Which was a pity, for he was doing very well and would probably have become stabilized.

Injections are always used in the beginning because it has been found from experience that oftentimes oral administration in the early stages is unsatisfactory. Presumably the drug is not absorbed. Even in some cases absorption from the intestine never becomes satisfactory. Some patients find that tablets do not keep them comfortable but that they are well if they have an injection once a week or so. Others can maintain themselves on tablets; others, again, seem to be able to discontinue treatment altogether, though whether permanently or not time will show. So far the longest known period of freedom from attacks without treatment is twenty months, remissions in this case having previously averaged three months and been getting shorter.

Other vasodilator drugs have been used, but none with such success as nicotinic acid. Administration of sodium nitrite intravenously has aborted attacks, for instance, but its side effects from fall of blood pressure are uncomfortable. Improvement was obtained in a few cases with glyceryl trinitrate, but no drug has proved so satisfactory as nicotinic acid. Whether its action in cases of this syndrome is that of a simple vasodilator or whether it acts also as a vitamin essential to the proper functioning of the autonomic system is not as yet determined. Of its efficacy in these cases in which there is vasoconstriction, however, there is no doubt, either in the minds of many patients or in my own.

Indiscriminate Use of Histamine.—A word must be said about the indiscriminate use of histamine in the treatment of this condition. The therapy has been advocated by Shelden and Horton[9] on the basis of

some spectacular results in producing relief from acute attacks. Unfortunately, it has not proved so effective in the long run in assuring relief from recurrence. I have reported recently 15 cases of patients who were insensitive to histamine and were treated with that drug, some by myself in error in the early days of this investigation and some by others according to the Mayo method.[11] In every case relapse, and in several instances actual deterioration, occurred, more or less soon after the beginning of the treatment, and it was a general experience that in such cases it was much more difficult to stabilize with nicotinic acid than in those in which histamine had not been given. Larger doses and more intensive and more prolonged treatment were required, and temporary setbacks, one or more, were almost the rule.

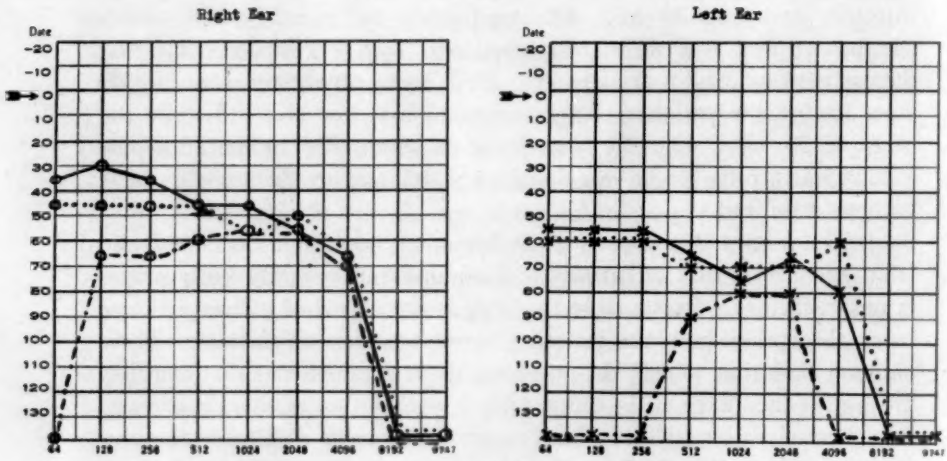


Fig. 2.—Audiogram in a case of Ménière's syndrome with insensitivity to histamine (primary vasoconstriction; patient treated first incorrectly with histamine). In this figure, and in figure 3, the solid line indicates hearing before treatment; the line of dots and dashes, hearing after mistaken treatment with histamine, and the line of dots, hearing after treatment with nicotinic acid (constrictor treatment).

The reason for the preliminary success and ultimate failure is not far to seek if the vascular basis for Ménière's syndrome is accepted. Histamine is a powerful vasodilator. Given intravenously by continuous drip, as advocated, it will by virtue of its vasodilator action rapidly relieve an attack caused by vasospasm. The immediate effect, indeed, is little short of miraculous. But the same thing can be done with any powerful vasodilator. Moreover, if the exhibition of histamine is repeated in these cases of vasospasticity, ultimately immunity to the drug is produced, vasodilatation fails to occur and attacks start again.

11. Atkinson, M.: Histamine in the Treatment of Ménière's Syndrome: An Appraisal, J. A. M. A. **119**:4 (May 2) 1942.

Necessity for Accurate Grouping.—Enough has been said to indicate the necessity for careful and accurate grouping of cases. As will be seen in the next section, the results when this is carried out are reasonably satisfactory. The results when it is not are very much the reverse. The effect of giving histamine indiscriminately in all cases has been discussed—it will benefit the small group of patients who are sensitive to histamine but may after the initial improvement do actual harm to the majority group of persons who are insensitive to the drug. In the same way, nicotinic acid administered wrongly to the patients who are sensitive to histamine will increase symptoms.

The deterioration produced by placing a case in the wrong group and giving the wrong type of treatment may appear graphically in an audiogram. Figure 2 shows the drop in hearing which took place when

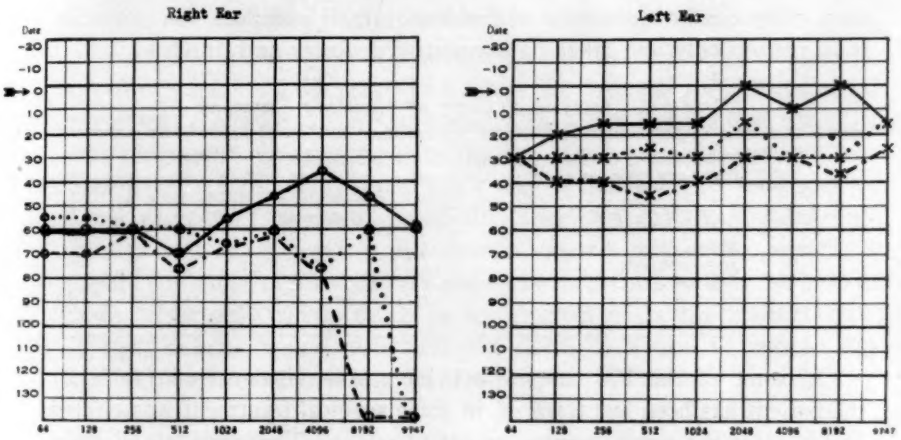


Fig. 3.—Audiogram in a case of Ménière's syndrome with sensitivity to histamine (primary vasodilatation; patient treated first incorrectly with nicotinic acid).

histamine was administered to a patient who was insensitive to histamine and the recovery when correct vasodilator treatment with nicotinic acid was adopted. Figure 3 shows a similar result when nicotinic acid was administered wrongly to a patient who was sensitive to histamine and the recovery which took place when the correct type of treatment was adopted.

RESULTS

In the assessment of the results of treatment of a condition such as this, which is subject to remissions even after long intervals of freedom from attacks, it is difficult to be both accurate and honest. Figures are uncompromising things—they draw in black and white, and here is a condition in which halftones are essential to a true picture. There are many extraneous or mitigating circumstances to be taken into account in making a just appraisal.

It would be easy to paint too rosy a picture, and because it is important not to do that, assessment of results has erred on the conservative side. Cure is not even considered; relief is regarded as the acme of success. Relief implies in this series that the case has been under observation for at least six months (no case included in these results has been studied for less and many have been observed for more, up to two years), and that there has been no attack for six months or since the most recent remission, whichever period is the greater. That is to say, no case has been included in which the patient has been seen for the first time since July 1941 (six months ago), and if the previous remission was, say, nine months ago, freedom from attack of any sort has been more than six months. Improvement implies continuing diminution in severity and frequency, or even cessation, of attacks, but for a period insufficient to qualify for the term "relief." Failure indicates relapse after an interval of freedom. In all instances, the reference is to vertigo only and takes no account of deafness and tinnitus.

TABLE 4.—*Analysis of Cases of Ménière's Syndrome*

Total number of cases (1940-1941)		108
Cases available for analysis		63
	Sensitivity to Histamine	Insensitivity to Histamine
Relief	12	20
Improvement	1	25
Relapse	1	4
	14	49

In some of the 108 cases which have been encountered (table 2) the patient has been lost track of, in some surgical treatment was elected and in some the results are too recent to assess. There are left 63 cases which are available for analysis at this time (Dec. 31, 1941).

Cases of Sensitivity to Histamine.—The results in this group have been eminently satisfactory, so much so that I have come to look on the putting of a case into this group as almost as much of a prognosis as a diagnosis. Of 14 cases available for analysis (table 4), there has been relief of attacks in 12 and of coincidental manifestations in many; in 1 case improvement continues; in 1 case only has a relapse occurred, and in that case there were many difficulties in the observation and treatment of the patient which led to his being lost track of.

Cases of Insensitivity to Histamine.—Results in this group have also been satisfactory, though not as satisfactory as those in the previous group. However, the figures do not tell all of the story.

Relief: Of 49 cases available for analysis (table 4), relief from attacks, which in some instances were very frequent, has been attained in 20. The relief in a few cases has been dramatic, as in that of a

woman of 53 who was having attacks in which she fell as often as two or three times a week, often hurting herself, and who after the first intravenous injection, eight months ago, has not had another attack. Usually, however, relief has been achieved only after prolonged treatment and carefully controlled dosage, not infrequently with one or two setbacks at first, against which the patient must be warned.

Improvement: Improvement has occurred in 25 cases. In some of these cases the patient has been free from attacks for weeks, even two or three months, and there are reasonable hopes that they may qualify for the higher rating shortly. In other cases the patients continue to have occasional momentary spells which do not bother them at all and which they can keep well controlled with oral medication. In yet others there are occasional more definite spells, which some patients can forestall largely or entirely with an injection. All the patients are sufficiently improved to be content with their lot.

Failure: Relapse to a condition as bad as or worse than it was previously has been the result in 4 cases. Of 2 cases in which the eighth nerve was sectioned, relief was obtained in 1, and in the other operation was too recent for assessment of results. In 2 cases the patients have been lost track of. This group has a way of increasing as time goes on, being recruited from members of the other two groups, and any such failures will be reported in due course. It is a matter for regret that papers on failure of a method are not more frequently seen in the medical press. Not only can so much be learnt from mistakes, but the report of such failures would also clear the decks of a deal of useless top-hamper that accumulates and hinders progress.

CONCLUSIONS AND SUMMARY

1. The importance of strictness in the definition of Ménière's syndrome is stressed.

2. The necessity for thorough examination, neurologic and general as well as otologic, in all cases is also stressed, and the danger of relying solely on the history and negative otologic findings is pointed out.

3. A method described previously of dividing cases of idiopathic Ménière's syndrome into two groups, those in which patients are sensitive to histamine and those in which they are insensitive, according to their reaction to an intradermal test, is briefly recapitulated.

4. The importance of such careful grouping of cases according to cause, in short of accurate diagnosis, is pointed out as an essential to success in treatment.

5. Further experience with treatment along such lines is recounted, and the results are evaluated.

123 East Sixty-First Street.

BILATERAL PARALYSIS OF ABDUCTOR MUSCLES OF THE LARYNX

REPORT ON SEVEN PATIENTS TREATED BY THE METHOD OUTLINED BY DR. BRIEN T. KING

LEWIS FRANCIS MORRISON, M.D.
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The loss of function of the abductor muscles of the larynx is a most serious and crippling condition. The fact that an adequate intake of air could be established by means of a tracheotomy tube was established many years ago. This procedure eliminated the necessity of working for each breath of air. It allowed the patient considerable latitude in regard to normal exercise but did not permit him to resume a wholly normal existence. The introduction of the valved tracheotomy tube was a step forward in that the defect could be disguised in a number of ways, but it did not relieve the patient of the worry, care and annoyance of a mechanical gadget.

A certain number of patients with bilateral posticus paralysis have existed without the advantage of an adequate intake of air, which could have been obtained by means of a tracheotomy tube. This does not detract from the seriousness of the condition, nor does it relieve the medical and surgical advisors of the responsibility of having added another burden to the community.

There is an opportunity to divide the cases of bilateral abductor paralysis into two main groups. The first group is composed of cases in which the onset is a gradual and progressive loss of function resulting from extralaryngeal insult either in the immediate neighborhood or from an intracranial lesion. The second group comprises those cases in which the onset is abrupt. The paralysis in this second group usually follows some acute infectious disease or the cutting of one or both recurrent laryngeal nerves during an operation in or about the thyroid area. There is a difference in the appearance of the vocal cords in these two groups. Several theories have been advanced to explain why the paralysis that is the result of a slowly progressive process should behave in the text-book manner, with the vocal cords in a cadaveric position, and why

From the Department of Surgery, Division of Otolaryngology, University of California Medical School.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1942.

the cords should be adducted when the onset is an abrupt one, such as is encountered after the acute infections and surgical severance of the recurrent laryngeal nerves. Thus far, none of the theories advanced offers a wholly satisfactory explanation for this difference.

The majority of cases of bilateral abductor paralysis seen at present are the result of some surgical procedure in or about the thyroid area, usually a second or third thyroidectomy. All clinic patients and a large percentage of the private patients entering the University of California Hospital for operation on the thyroid are examined prior to operation by both the surgical staff and some member of the department of otorhinolaryngology. Separate notations of the laryngeal condition are made by both services. The incidence of unilateral abductor paralysis in patients who have had one or more thyroidectomies and are now admitted for reoperation is high. The possibility of injury of the functioning recurrent laryngeal nerve during the impending operation is an ever present hazard, due to the increased difficulties attending all secondary and tertiary operative procedures. Therefore it is important to know the exact condition of the laryngeal musculature prior to surgical intervention.

Patients suffering with bilateral abductor paralysis should be further classified as to whether they are tracheotomized or nontracheotomized. The tracheotomized group has an adequate air intake, but the patients are most anxious to get rid of the tracheotomy tube. They soon forget the great relief the tube afforded when it furnished an adequate and effortless air intake. The annoyance of the tube, its demand for constant attention and the psychic trauma attending the ever present necessity of explaining to the curious why it is essential tend to outweigh the advantages of even a most efficient valved tube. The second group, composed of patients who do not have the discomfort of a tracheotomy tube or the advantage of an adequate air intake, comprise a sorry and pathetic group. Their world is practically a ground floor plan. Stairs, in any number, are an almost unsurmountable obstacle. They learn by sad experience that they must neither hurry nor become excited. Their narrowed world is filled with fear that something will occur to make their breathing still more difficult. An acute infection of the respiratory tract and associated laryngitis are too frequently the answer to their fears. The fact that in a few scattered instances complete recovery of function occurred as long as two months after either some surgical procedure or an acute infection may explain why, during the first week or so, a medical or surgical advisor encouraged the patient to carry on for a while longer. One fact that undoubtedly encouraged the advisor to hope with the patient, or to hearten the patient to hope

with him, was that until Dr. Brien T. King [1] paved the way, there was no corrective measure that offered reasonable assurance of both an adequate airway and a usable voice.

Contributions that can be considered as milestones in laryngology are not presented often. Such a contribution has been made recently. The preliminary report published by Dr. King probably received little more attention in most quarters than it did here. The main interest was expressed by those men who do most of the thyroid surgery and are in intimate contact with the complications of this operation. The otolaryngologists showed little enthusiasm, and the attitude in general was that it was just another bit of surgical technic. The memories of the corrective measures proposed in the past were not especially inspiring. In general, these measures were frank failures. Hoover [2] stated that by employing a submucous cordectomy he was able to obtain beneficial results in three fourths of his patients. The good results consisted in establishing a functioning airway at the expense of a useful voice. This was a step forward but did not offer enough to satisfy either the patient or the surgeon.

Nearly a year after the publication of his preliminary report, Dr. King was induced to come to the University of California Hospital and demonstrate his operative procedure. A series of 4 patients were operated on at that time. I was fortunate in being invited to watch the procedure from a vantage point. Fortune further favored by permitting ample opportunity to discuss with him the various steps of the operation. His interest and sincerity were inspiring. His expenditure of time and experience and his patience in going over the details were most generous. All of this may seem a bit out of place in a report of this type, but I feel that he has made a definite contribution and is therefore entitled to all possible praise. The results he obtained are not due to accidental stumbling onto something of value. He had a definite idea and then went to work on the problem in a sound manner. This entailed a review of the literature and many hours of study of the embryology, development, function, anatomy and physiology of the larynx. In addition, he familiarized himself with the use of the head and larynx mirrors, so that he could examine a larynx by the indirect method. He spent ample time in working with unfixed anatomic specimens to establish the soundness of both the idea and its practical application. He was emphatic about the necessity of reviewing the anatomy

1. King, B. T.: A New and Function-Restoring Operation for Bilateral Abductor Cord Paralysis: Preliminary Report, J. A. M. A. **112**:814 (March 4) 1939.

2. Hoover, W. B.: Bilateral Abductor Paralysis: Operative Treatment by Submucous Resection of the Vocal Cords, Arch. Otolaryng. **15**:339-355 (March) 1932.

of the area and the use of unfixed material for perfecting technic. I can now concur with him on this subject. For six months after his visit I exploited every available source of unfixed material. The energy spent in obtaining such specimens and the hours spent in going over the surgical steps on this material are of untold value. No matter how well one may think one knows the anatomy of this area, the operation should not be undertaken with a living patient until the various steps have been worked out in detail a number of times on the unfixed cadaver. One further fact should be given consideration prior to operating on a living subject. A variable portion of the field of operation will not be through virgin territory. In this area of previous operation the anatomic landmarks are often slightly obscure, misplaced, or even absent, as a result of the previous surgical procedure and the mature scar tissue, which does not necessarily follow the normal cleavage planes.

The various steps of the procedure have been well outlined by Dr. King. The matter of the initial incision being laid down parallel to and along the border of the sternocleidomastoid muscle was discussed with him. It did seem that a modified Kocher incision would both allow better exposure of the anatomic landmarks and result in a less disfiguring scar. He agreed that the suggestion was in order and said that he had given it some consideration, but had not done anything about it as yet because he was more comfortable following his original procedure. I have used the modified Kocher incision throughout and find that it offers definite advantages. It was interesting to read that Kelly [3] had arrived at a similar conclusion.

The selection of the anesthetic agent is a problem that must be solved by the individual surgeon after he has given due consideration to the advantages and disadvantages of the various available agents as related to, first, the patient, and, second, the ability of the anesthetist to control the patient with the selected type. Dr. King reports good results with the intravenous use of pentothal sodium (sodium ethyl-[1-methylbutyl]-thiobarbiturate). Dr. Kelly obtained good results with intratracheal anesthesia in 1 case and local anesthesia in another. Intravenous administration of an anesthetic has the drawback of the drug being entirely out of control once it has entered the blood stream. The orthodox method of administering an anesthetic intratracheally by means of a rubber tube through the larynx does not permit the examination of the vocal cords during the operative procedure. There are times when such an examination is both a comfort to the surgeon and of vital importance to the patient.

3. Kelly, J. D.: Surgical Treatment of Bilateral Paralysis of the Abductor Muscles, Arch. Otolaryng. **33**:293-304 (Feb.) 1941.

In the series of cases presented here basal anesthesia was induced in all the patients by avertin with amylene hydrate (not less than 80 or more than 90 mg. of avertin per kilogram of body weight) and a mixture of nitrous oxide and oxygen introduced through the tracheotomy opening. This can be accomplished by means of a rubber tube that fits the outer tracheotomy tube snugly or by means of a rubber tube introduced into the trachea directly through the tracheotomy opening. Even though a closed system can be obtained and maintained by the latter method, I have not felt justified in using either the cutting or the coagulating current during the operation. The few additional minutes that are necessary to obtain hemostasis by clamping and tying the individual vessels does not add to the operative risk. Usually the nitrous oxide and oxygen mixture does not give sufficient relaxation to permit laryngoscopy without considerable reaction from the patient. In order to obtain the neces-

Surgical Treatment of Bilateral Paralysis of the Abductor Muscles of the Larynx

Case number	1	2	3	4	5	6	7
Sex	Female	Female	Female	Female	Female	Female	Female
Age	28 yr.	27 yr.	40 yr.	44 yr.	39 yr.	38 yr.	35 yr.
Duration of paralysis	2 yr.	2½ yr.	9 yr.	3 yr.	2 days	20 yr.	2 yr.
Thyroidectomy, time	(1) 1938 (2) 1939	1938	(1) 1930 (2) 1932	(1) 1937 (2) 1938	1941	1921	1939
Side of operation: Left	2/28/41	3/20/41	4/1/41	1/12/41		6/13/41	7/16/41
Right	4/30/41			6/ 9/41	6/12/41		
Voice prior to operation	Fair	Good	Good	Fair	?	Good	Good
Voice after operation	Fair	Better	Good	Fair	Fair	Good	Good
Resultant airway	Good	Good	Good	Good	Good	Good	Good
Tracheotomy tube removed	6/9/41	4/16/41	4/16/41	6/17/41	7/30/41	7/9/41	7/24/41
Tracheotomy scar removed	7/9/41	7/23/41	7/8/41	9/11/41	None	None	None

sary relaxation, cyclopropane is used in place of the nitrous oxide for a few minutes prior to and during examination of the larynx. There have been no untoward reactions to this anesthetic either during or after the operation.

The results obtained, as outlined in the table, show that when properly carried out the procedure is a satisfactory one in that both an adequate airway and a usable voice may be expected. It is evident that the procedure is neither so difficult nor so involved that satisfactory results can be obtained only by its originator. It is practical for use by any one who will take the time to learn or review, as the case may be, the essential anatomy of the area prior to attempting the operation on a living subject and who has at least average conception of the few simple mechanical problems attending such a surgical procedure. The surgeon should feel free and comfortable in using either the left or the right side for the approach. There are instances in which one side will offer better possibilities than the other for an excellent rather than a

mediocre result. In other instances it may be necessary to operate on one side first and on the other at a later date. This problem presented itself in cases 1 and 4 in the present series. The operation and its results in cases 1, 4 and 5 are of sufficient interest to warrant a more detailed report than can be obtained from the table.

REPORT OF CASES

CASE 1.—Operation on the left side was performed on Feb. 28, 1941. The actual mechanics of the procedure progressed in an orderly manner. Direct laryngoscopic examination during the operation showed that tension on the sutures around the left arytenoid cartilage pulled the cord well over to the left side and produced an adequate airway. These observations were checked after the left arytenoid cartilage was made fast to the thyroid cartilage and were checked again after the omohyoid muscle was attached to the arytenoid cartilage. The larynx was examined by the indirect method on the fourth postoperative day, when, in spite of a moderate amount of edema of the left arytenoid area, it was evident that the left cord was well over from the midline. Closure of the tracheotomy tube for a few moments showed that the airway was adequate. Postoperative notes made on subsequent days showed that there was diminution in available airway in spitie of regression of the edema of the left arytenoid area. Examination with the larynx mirror gave the impression that the left vocal cord and arytenoid cartilage were slowly approaching their former position at the midline. By the end of the third postoperative week it was evident that, although the airway was slightly improved as compared with the available space prior to operation, it was not sufficient to permit removal of the tracheotomy tube. This was disappointing. The explanation for the apparent failure that first came to mind was either that the suture fixing the arytenoid cartilage to the lateral wall of the thyroid cartilage had been insecure or that it had eroded its way through either the arytenoid or the thyroid cartilage and in so doing had allowed the cord to resume its former position. Examination of the larynx a few days later resulted in a true explanation of the difficulty. It was then realized that there had been no technical error. The chink of the glottis was seen to be shifted to the left about 30 degrees from the midline posteriorly, and the right cord had migrated or been pulled past the midline over to the left cord. To be sure, this did not change the immediate condition of the patient, but it improved the mental status of the operator.

Two months after the first operation the right side was corrected. The interarytenoideus muscle was found to be thick and well developed. At this procedure, the muscle was completely detached from the right arytenoid cartilage, and the cartilage was fixed to the thyroid cartilage with a silk suture of medium weight. Prior to the second operation, the patient was assured that an adequate airway would be obtained and that after a short period for recovery the tracheotomy tube would be removed. However, the fact was emphasized that in order to obtain this result it might, and probably would, be necessary to sacrifice the thought of a wholly usable voice. The patient was agreeable to this result. However, the worst did not materialize. The air intake is more than adequate. The tracheotomy tube has been removed, and the patient is both pleased and grateful. Her voice is not to be classed with the most pleasant in that it is a bit on the husky side, but it has fair quality and excellent volume, as any one will ascertain if he happens to be anywhere in her general vicinity. The voice is usable and is being used.

CASE 4.—The original operation on the left side was performed by Dr. Brien T. King. After the sutures around the arytenoid cartilage had been tied, he examined the larynx by the direct method and concluded that the displacement to the left was too great. The suture fixing the arytenoid to the thyroid cartilage was cut. This was done with the thought that the omohyoid transplant would offer sufficient pull to maintain an adequate airway, which, however, was not obtained. Prior to attempting to establish an adequate airway through the larynx, I discussed the problem with Dr. King and seriously considered reoperating on the left side. This might have allowed an opportunity to observe whether, how well and where the omohyoid muscle was attached. However, when the day of operation arrived, some five months after the first procedure, the idea of having to fight scar tissue the entire time dampened the scientific ardor. The operation was done on the right, or opposite, side. The resulting airway is adequate, and the tracheotomy tube has been removed. The resulting voice is only fair but is no worse than it was prior to the first operation.

CASE 5.—The patient presented a most interesting problem. Operation on the thyroid, done on June 10, 1941, proved to be a difficult procedure. The gland was large, very firm and adherent to everything in the surrounding territory. Bleeding was profuse and difficult to control. The thyroid surgeons suspected that the tissue was malignant. Their suspicions were confirmed both by frozen sections obtained during the time of operation and by paraffin material run through by the rapid method and cut, stained and examined the following day. During the latter part of the operation on the thyroid the patient showed evidence of respiratory difficulty. This difficulty was only partly relieved when the patient was put in an oxygen tent. On the following day, June 11, a tracheotomy was performed. The established facts were bilateral paralysis of the abductor muscles and the malignant condition. There was much discussion as to what the next step should be and when it should be undertaken. Finally, it was agreed that the ideal time for the King operation was the present. If the patient was to receive radiation therapy prior to establishing an adequate airway, the latter procedure would have to be delayed for several months, and, if attempted then, the hazards attending surgical intervention in a heavily radiated field would demand consideration. Although immediate correction meant entering the area of operation for the third time and on the third consecutive day, the risk involved was not considered inordinate. From the operator's standpoint, such an early approach was an easy one. The original thyroid incision was used. The major portion of the dissection had been done by the thyroid surgeons. There was little bleeding; the exposure was more than adequate, and since it was too early for the formation of either granulation or scar tissue, the anatomic structures and landmarks were easily discernible. The incision was closed in the usual manner except for a small area in the midline of sufficient size to permit the presence of the tracheotomy tube. The postoperative course was a bit stormy. There was mild pneumonia, and the field of operation showed early signs of infection. Both conditions subsided in due time and produced no unpleasant sequelae. The result is excellent as to airway, and the patient has a wholly useful voice. The tonal qualities are open to some discussion, owing to the fact that the patient has been without sound perception since childhood and when she does speak, she uses a high-pitched monotone.

CONCLUSIONS

The results of eight operations on 7 patients suffering with bilateral paralysis of the abductor muscles of the larynx are presented.

The King operation is technically and actually a successful procedure. It is not a one man operation and can be done by any one who will spend the time necessary to acquire a working knowledge of the regional anatomy and the function of the part involved.

The King operation fulfils all its promises. Both an adequate airway and a usable voice are obtained. It has no restrictions as to age and is not limited by either the cause or the duration of the paralysis. There are variations in the technical difficulties, but they are no greater than the surgeon should expect and be equipped to surmount.

All should feel grateful to Dr. Brien T. King for having presented this new and function-restoring operation for bilateral paralysis of the abductor muscles.

Dr. Brien T. King assisted me in equipping myself to carry out the procedure.

490 Post Street.

NEURINOMA OF THE FACIAL NERVE IN THE MIDDLE EAR AND MASTOID

REPORT OF A CASE

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Neurinoma of the facial nerve has received scant mention in the literature and is apparently a rare entity.

The discovery of such a tumor at about the time Williams and Pastore [1] reported a case from the Mayo Clinic stating that "careful search of the literature has failed to reveal any report of a similar case" prompted a further search of the literature and a report of another case.

GENERAL SURVEY

Until Virchow's treatise on tumors (1863-1867) [2] all tumors of nerves were classed indiscriminately as neuromas. He subdivided these into true and false neuromas, the false neuromas composed chiefly of connective tissue being classified as neurofibromas.

In 1870 von Bruns [3] described plexiform neurofibroma, but wide attention was not called to this type of tumor until the classic work of von Recklinghausen [4] on neurofibromatosis in 1882. This disorder, universally known as Recklinghausen's disease, is characterized by the appearance of multiple tumors of subcutaneous and other nerves and by areas of pigmentation of the skin, varying in hue from light to dark brown.

Microscopic examination of the lesions of neurofibromatosis shows that the tumors arise from the connective tissue of the nerves and that

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From the Department of Otolaryngology, University of Southern California School of Medicine.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., Oct. 30, 1941.

1. Williams, H. L., and Pastore, P. N.: Neurofibroma of the Facial Nerve in the Facial Canal with Destruction of the Labyrinth and Mastoid Process, Arch. Otolaryng. **29**:977-981 (June) 1939.

2. Virchow, R.: Die krankhaften Geschwülste, Berlin, A. Hirschwald, 1863-1867.

3. von Bruns, P., cited by Garrison, F. H.: An Introduction to the History of Medicine, ed. 2, Philadelphia, W. B. Saunders Company, 1917.

4. von Recklinghausen, F. D., cited by Garrison, F. H.: An Introduction to the History of Medicine, ed. 2, Philadelphia, W. B. Saunders Company, 1917.

they are accordingly simple fibromas through or around which the nerve fibers pass.[5]

Thompson,[6] in 1903, divided neurofibromas into two types, the multiple false neuromas being those tumors which are found only in Recklinghausen's disease and the solitary false neuromas arising from the connective tissue of nerves.

While earlier writers expressed the belief that these growths originated in the sheath of Schwann, more recent observers have expressed the opinion that the lesions are not fibromas but a product of the proliferation of Schwann's cells or analogous elements. For this reason the term neurinoma, i. e., tumor of a nerve fiber, was proposed by Verocay,[7] in 1910, as describing the condition more accurately.

This term has been the one most commonly used in the literature reviewed, although similar tumors elsewhere have been described as neuroma, neurofibroma, schwannoma, perineural fibroblastoma, neurilemmoma and peripheral glioma.

Such a tumor arising from the auditory nerve has been described as acoustic neuroma or tumor of the cerebellopontile angle. Of the cranial nerves the auditory is the most frequently involved, and according to Dandy,[8] "Cerebellopontine tumors are perhaps the most common of the benign, encapsulated tumors of the brain."

Reports of involvement of the optic nerve tract are not uncommon, although in many cases such involvement has been associated with Recklinghausen's disease.

It was not until 1927 that a tumor of a purely motor cranial nerve was reported by Kovacs[9] as a "solitary neurinoma of the oculomotor nerve." A year later Schröder[10] reported a case of "neurinoma of the accessory and hypoglossus."

GENERAL PATHOLOGIC CHANGES

Solitary neurinomas are classified as benign tumors. However, as they increase in size they tend to invade adjacent tissues and may even destroy vital structures and in this sense may become clinically malignant. They usually grow rather slowly, and considerable time may elapse before this takes place. They are often firm and well encapsu-

5. Delafield, F., and Prudden, T. M.: A Textbook of Pathology, ed. 13, New York, William Wood & Company, 1924.

6. Thompson, cited by Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 968.

7. Verocay, J.: Zur Kenntnis der "Neurofibrome," Beitr. z. path. Anat. u. z. allg. Path. **48**:1-69, 1910.

8. Dandy, W., in Lewis, D.: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc., 1932, vol. 12, chap. 1, p. 534.

9. Kovacs, cited by Schröder[13] and Altmann.[15]

10. Schröder, cited by Schröder[13] and Altmann.[15]

lated, thus facilitating complete removal, even intracranially, as in the case of the acoustic neuromas.[8] Even when complete removal is not attained recurrence is usually slow. At other times the tumor is soft and even cystic and degenerative changes may occur. It is difficult to remove such a tumor completely. It is possible that the less fibrous types may be more susceptible to irradiation. At times these tumors may undergo periods of spontaneous arrest and even regression, so that history and clinical findings are often confusing.

MICROSCOPIC ANATOMY

Gnassi and Borrone [11] have presented a description of the microscopic anatomy of these tumors which is so complete that I have taken the liberty of quoting from them as follows:

> According to the well accepted classification of Antoni, there are two types of tissue.
>
> TYPE A.—The most conspicuous feature is palisading of the nuclei . . ., which may also be arranged in eddies or in fasciculi . . . or which may form whorls. The nuclei are elongated, slender and sometimes curved and contain a loose chromatin material. The nucleoli may be seen in the degenerating areas. Arranged at each end of the nucleus is a scanty branching, anastomosing cytoplasm which tends to form a syncytium. The outline of the cells is difficult to make out. Silver impregnation reveals straight long wiry, sometimes wavy reticulin fibers running parallel to the long axis of the nucleus. In the degenerate portions these fibers tend to lose their arrangement and may be decreased or absent, and in other places they may be devoid of cellular elements. The fibers are not in broad sheets or wrapped around the cells, as in a fibroblastic tumor. Cylinder-like structures, resembling Wagner-Meissner tactile corpuscles, may be seen, as pointed out by Masson.
>
> TYPE B.—This is a degeneration of type A . . . and is often associated with it in various proportions. The cells are widely separated and their protoplasm tends to form a reticulum-like structure. The reticulin fibers have a chaotic arrangement. The ground substance has a loose texture. Rounded cystic spaces, fluid collections of degenerated tissue, constitute this so-called microcystic degeneration. Collagen fibers are rarely seen. The tumors are rather vascular, and hyalinization of the walls of the blood vessels is sometimes seen.

REVIEW OF THE LITERATURE ON NEURINOMA OF THE FACIAL NERVE

The first case of neurinoma of the facial nerve to be found described in the literature was reported by Schmid,[12] in 1930. The patient was a 16 year old girl who had a facial paralysis of indefinite duration, and a

11. Gnassi, A. M., and Borrone, M.: Schwannoma, Arch. Otolaryng. **27**:766-772 (June) 1938.

12. Schmid, C.: Neurinom des N. facialis, Jahresversammlung der Gesellschaft Schweizer Hals- und Ohrenarzte, Zurich, July 1930; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **16**:329, 1930-1931.

smooth tumor mass was seen on the posterior wall of the external auditory canal. The tympanic membrane and the hearing were normal, and the mastoid appeared normal. Exploration of the mastoid revealed a cavity "the size of a bean," filled with granulation-like tumor masses. In the bottom of the cavity the facial nerve lay exposed in the descending portion of its bony canal. The tumor masses were removed, and histologic examination led to a diagnosis of neurinoma originating from the sheath of the facial nerve. The postauricular wound healed promptly, and there was some improvement in the facial paralysis.

Schröder,[13] in 1931, reported the second case, that of a 25 year old farm hand with a history of paralysis of the right side of the face for eight years and chronic purulent otitis media for four years. He had undergone two mastoid operations, and at the second operation tissue was obtained which was found microscopically to be neurinoma. There was complete loss of hearing in the right ear, but he had never experienced any vertigo. Caloric tests could not be made because of obstruction of the auditory canal by the tumor. At the third operation a large tumor mass was found which completely filled the middle ear and had destroyed the posterior wall of the external auditory canal and a large part of the mastoid process. It had also invaded the middle cranial fossa, so that complete removal of the tumor was impossible. Irradiation was employed postoperatively. One year later the operative wound had healed with the exception of a small fistula, which was demonstrated by injection of iodized poppyseed oil to extend into the frontal lobe of the brain. The patient felt perfectly well and was able to resume his work, but the deafness and facial paralysis persisted.

The third case was reported by Grossmann and Leidler[14] in 1932 before the Austrian Otologic Society and was later reported in greater detail by Altmann[15] in his series of 4 cases from the University Clinic in Vienna. The patient had first been seen in 1926 at the age of 20, with a history of paralysis of the right side of the face for eighteen months. The external auditory meatus was completely filled with a large polyp which arose from the posterior canal wall. There was deafness of the conductive type. Vestibular responses were normal, as were roentgenograms of the mastoid. Biopsy gave the impression of leiomyoma, and the patient was treated with radium. The tumor receded slightly, and a year later a normal tympanic membrane could be seen and hearing had improved somewhat. Six years later the patient returned with facial paralysis unchanged and increased deafness and headache. Roentgen examination revealed an erosion of the posterior

13. Schröder, C.: A Case of Neurinoma of the Facial Nerve, Hospitalstid. **74**: 56-60, 1931.

14. Grossmann, B., and Leidler, R.: Drei Tumoren des Ohres: Fall I, Monatschr. f. Ohrenh. **67**:353-357, 1933.

15. Altmann, F.: Ueber neurogene Tumoren des absteigenden Fazialisteiles, Monatschr. f. Ohrenh. **69**:1032-1048, 1935.

wall of the external auditory canal but normal pneumatization of the mastoid cells. Operation revealed a solid tumor occupying the middle ear and mastoid antrum with destruction of the posterior wall of the auditory canal and the facial canal in its descending portion. Histologic examination proved the tumor to be a neurinoma. Recovery was uneventful, but the facial paralysis persisted.

In 1935 Altmann [15] published the first comprehensive article on these tumors, establishing them as a definite clinical entity and reporting 3 additional cases from his own clinic, in addition to a detailed review of the 3 previously mentioned cases. His first case was that of a 26 year old woman seen in 1932 with paralysis of the right side of the face and a polypoid tumor mass filling the right external auditory canal. There was deafness of the conductive type. Roentgenograms of the mastoid were normal. Repeated biopsies resulted in a diagnosis of neurinoma, and irradiation by the Coutard method was employed. Two years later the symptoms and findings were essentially unchanged. His second and third patients both came to operation because of chronic aural discharge and signs of labyrinthine involvement. Both had facial paralysis preceding the symptoms in the middle ear by several years. Roentgen examination showed clouding of the mastoid cells. At operation there were destruction of mastoid cells and suppuration extending into the perilabyrinthine cells, in addition to the tumor masses which were histologically proved to be neurinomas. Recovery was uneventful with the exception of a permanently dead labyrinth in the third case, and persistent facial paralysis in the second and third cases.

Greifenstein,[16] in 1936, added a seventh case, in which the tumor was discovered post mortem in a patient who died after an operation for removal of a hypophysial tumor. Previous clinical examinations had revealed no facial paralysis and only slight impairment of hearing. When the temporal bone was sectioned a neurinoma was found in the niche of the oval window, and its origin was definitely traced to the horizontal portion of the facial nerve. Greifenstein concluded that clinical symptoms of the impairment of the facial nerve appear only after the neurinoma has reached a considerable size, that disturbances in the conduction of sound likewise occur rather late and that erosion of the bone develops only if there is no longer room for expansive growth of the tumor.

In May 1937 Ohnishi [17] reported a case in which there was the usual history of facial paralysis preceding a chronic otitis media. At operation a tumor was found arising from the descending portion of the facial nerve. The histopathologic diagnosis was neurinoma. Recovery was uneventful with the exception of persistent facial paralysis.

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16. Greifenstein, A.: Zur Kenntnis der isolierten Facialisneurinome, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **142**:50-53, 1936.

17. Ohnishi, Y.: A Case of Neurinoma of the Facial Nerve with Facial Paralysis, Oto-rhino-laryng. **10**:420-425, 1937.

In November of the same year Altmann's second article [18] appeared, in which he added another case of his own to the 6 he had previously collected. He also reviewed Greifenstein's case but apparently was not aware of Ohnishi's report at that time.

In June 1939 Williams and Pastore [1] added a tenth case and made the first contribution to the American literature with their report of a neurofibroma of the facial nerve arising from the horizontal portion of the facial canal, with invasion of the mastoid and destruction of the labyrinth. They suggested that when specimens are not obtainable from the aural canal or when the results of biopsy are inconclusive radical mastoidectomy is indicated to make a differential diagnosis and to remove a nonmalignant tumor if it is present.

In September of the same year Rejto [19] reported another case, in which the site of origin of the tumor was definitely determined to be the horizontal portion of the facial nerve. The histopathologic diagnosis was neurinoma, and the patient made an uneventful recovery except for having persistent facial paralysis. Rejto confessed that such a tumor was not suspected prior to the operation, which was done solely because of chronic aural suppuration. He stated that the history of facial paralysis preceding aural symptoms was disregarded because of the frequent unreliability of patients' statements concerning the onset of a chronic otitis.

The twelfth case in the literature prior to the present writing was reported by Vysotskaya [20] in 1940. I have been unable to review this article, but the title suggests that it belongs in this group.

COMMENT

The striking similarity of these case reports confirms the observations of Altmann,[15] in 1935, that neurinoma of the facial nerve, although rare, is a definite clinical entity. His conclusions on the basis of the first 6 cases have merely been reiterated by subsequent observers. In all cases but the one in which the condition was discovered post mortem [16] the first symptom was facial paralysis, which persisted during the entire course of the disease. This paralysis may for a long time be the sole symptom. Later, if the ear is examined, a tumor mass, appearing to arise from the posterior canal wall, may be seen in the cases in which the tumor originates in the descending portion of the nerve. In the cases in which the tumor arises from the horizontal portion of the nerve, invasion of the middle ear with destruction of the tympanic membrane must occur before the tumor becomes visible in the external auditory canal. The

18. Altmann, F.: Zur Kenntnis der Tumoren des absteigenden Fazialisteiles, Monatschr. f. Ohrenh. **71**:1287-1292, 1937.

19. Rejto, A.: Fall eines vom Gesichtsnerven ausgehenden Tumors, Monatschr. f. Ohrenh. **73**:615-618, 1939.

20. Vysotskaya, L. G.: Case of Neurinoma of Facial Nerve, Zuhr. ush., nos. i gorl. bolez. **17**:235-236, 1940.

impairment of hearing may be due to mechanical obstruction or chronic otitis media, which ensues as a result of secondary infection. The latter frequently obscures the diagnosis, especially if the mastoid cells become involved. The growth of the tumor is slowly expansive, and because of its position in the middle ear there are attendant dangers of intracranial complications.

In making the differential diagnosis one has to consider chronic infection of the middle ear, chronic mastoid suppuration, tuberculosis and carcinoma. Careful history taking and roentgen examination should help to rule these out. Occasionally biopsy is helpful. The history of facial paralysis preceding aural symptoms is of the utmost importance. When results of repeated biopsies and roentgen examinations are inconclusive, exploratory operation is indicated, consideration being given to the risk of surgical intervention as opposed to that of the disease at the time the decision is to be made.

In the case to be reported the question of tumor arose, but owing to the fact that neurinoma of the facial nerve had not yet appeared in the American literature and the previous cases reported were unknown to me, the diagnosis was missed and radical mastoidectomy was advised solely on the basis of a chronic mastoiditis.

REPORT OF CASE

R. H., a white man aged 50, was first seen Oct. 11, 1938, complaining of a discharging right ear for fifteen years, deafness for twenty years and paralysis of the right side of the face for thirty years. He had rarely had any pain or headache and had never experienced any vertigo suggestive of labyrinthine disturbance.

His past history included typhoid, malaria, gonorrhea and hay fever.

Physical examination revealed a well developed, well nourished man with complete paralysis of the right side of the face of the peripheral type. The right side of the forehead and the brow could not be wrinkled. The right eye could not be closed, and the lower lid sagged, as did the muscles of the face, which were so flaccid that the right cheek ballooned out when he talked. The eyes showed no spontaneous nystagmus, and the pupils were equal and reacted normally. The right ear was completely deaf, and a thin, serosanguinous, foul-smelling discharge came from the external canal. Only a small remnant of tympanic membrane was seen, and red granulations filled the middle ear. There was no edema over the mastoid, and deep pressure elicited no tenderness. The left ear was normal. The results of the rest of the physical examination were entirely normal, including a careful neurologic examination. A blood count and the urine were normal. The Wassermann and Kahn reactions were negative.

A specimen for biopsy was taken from the tissue in the middle ear and reported to be chronic inflammatory granulation tissue.

Roentgenograms of the mastoids were taken and the following observations made: The left mastoid appeared normal. On the right side there was a considerable amount of pneumatization, but it was not so marked as on the left. The entire petrous portion, as well as part of the mastoid process, showed dense sclerotic bone, and the remaining air spaces were filled with cloudy density. The middle

ear and the external auditory canal appeared considerably enlarged by some circumscribed erosive process.

The possibility of a neoplasm causing this erosion was considered, but surgical therapy was undertaken solely on the basis of a diagnosis of chronic mastoiditis.

On November 2, with the patient under general anesthesia, the usual postauricular exposure of the mastoid was made. The dense, sclerotic external plate was removed with a gouge, and immediately a large, smooth, bulging mass presented, not unlike a herniation of the lateral sinus. However, it proved to be firm and presented a free border inferiorly. This was followed anteriorly, and it was found that most of the posterior bony wall of the external auditory canal was eroded and the mass extended into the hypotympanum, which was considerably enlarged, as was the region about the eustachian tube. The tumor filled the middle ear, aditus ad antrum and mastoid cavity, and it seemed firmly attached superiorly in the region of the tegmen tympani. It was carefully shelled out by blunt dissection, beginning in the hypotympanum and tubal region, but as the shelling-out

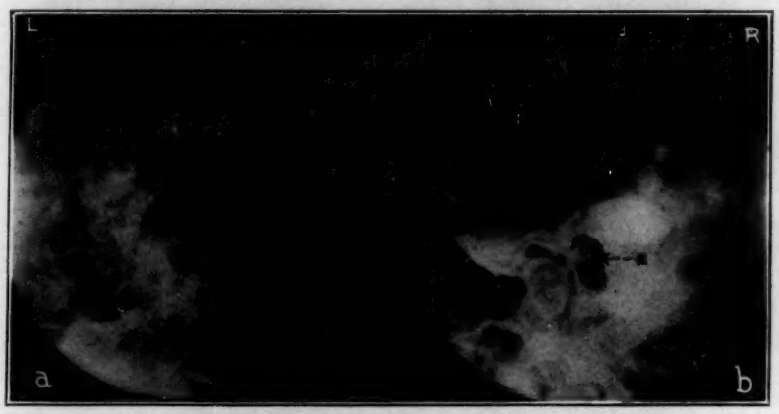

Fig. 1.—Roentgenograms of (*a*) the normal left mastoid and (*b*) the right mastoid, showing erosion of the middle ear and external canal (arrow).

process was extended posterosuperiorly it became apparent that it was adherent to the mesial wall of the middle ear, in the region of the horizontal portion of the facial canal, and the overlying exposed dura of the middle cranial fossa. In view of adhesions to the dura, this line of procedure was discontinued, and the tumor was approached posteroinferiorly in the mastoid cavity. It continued to shell out easily anterosuperiorly until it was entirely free except for its attachment to the superior portion of the mesial wall of the middle ear and the overlying dura. Adhesions to the dura were carefully separated until only the region of the facial canal remained, and this was freed by sharp dissection, a small stump of dense fibrous tissue being left overlying the horizontal portion of the facial nerve. Care was taken not to disturb the stapes, but the other ossicles were missing. The tumor was removed intact and measured 2 by 1.5 by 1.5 cm.

A small piece of the tumor near its attachment and some bits of tissue from the mastoid cavity were sent to the laboratory for immediate frozen section and report before the wound was closed. The pathologic diagnosis was fibroma with nerve elements and chronic inflammatory granulation tissue. Because of this it was decided that implantation of radium was not indicated.

Fig. 2.—Appearance after exposure of the mastoid cavity.

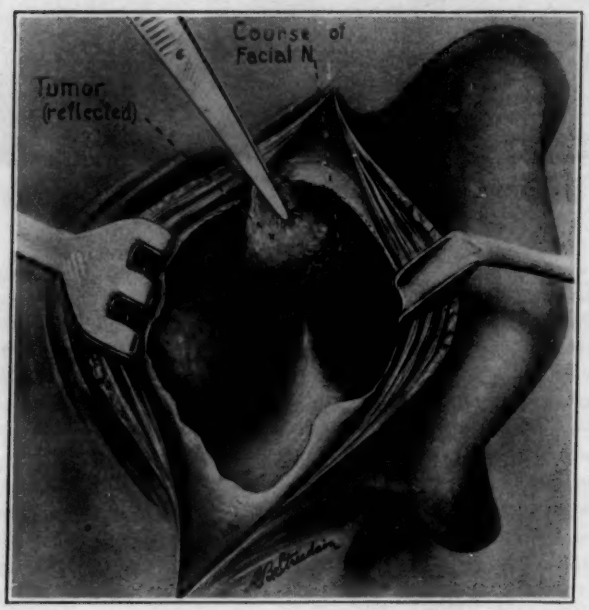

Fig. 3.—Tumor freed, except for its attachment to the horizontal portion of the facial canal.

The remaining cavity was practically a completed radical mastoidectomy, and very little additional work was necessary. The usual plastic flap and closure of the postauricular incision were carried out, and the cavity was packed through the external auditory canal.

After paraffin sections of the main tumor mass had been prepared and studied, the following report was made by Dr. E. M. Hall, professor of pathology at the University of Southern California:

"On gross examination the specimen consisted of a roughly pyramidal-shaped gray-white tumor measuring approximately 2 by 1.5 by 1.5 cm. In addition, there were several small fragments measuring each about 5 mm. in diameter. The tumor appeared to be encapsulated.

"Microscopic examination showed that the greater part of the section consisted of whorls and strands of rather dense partly hyalinated fibrous tissue. Roughly

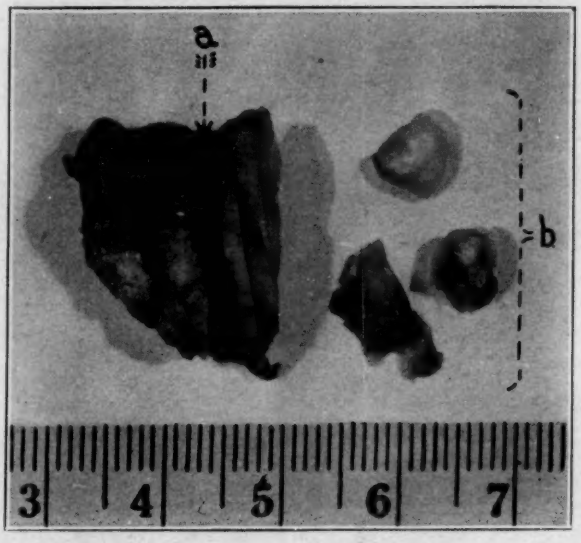

Fig. 4.—Photograph of the tumor after fixation, showing (*a*) a block removed for paraffin section and (*b*) granulation tissue and fragment of the tumor removed for frozen section.

one third of the section was more cellular, consisting of elongated fusiform cells arranged in whorls. There was moderate palisading of the nuclei. These areas were basophilic in their staining with hematoxylin and eosin, and the areas resembled nerve sheath cells. Sections stained with phosphotungstic acid revealed blue-staining nuclei and fibrils in the cellular areas, while in the fibrous areas the fibrils stained reddish brown. In places the surface was covered by stratified squamous epithelium which varied considerably in thickness. There was some proliferation of the epithelial pegs in the thicker areas. There was some calcified osteoid tissue in one of the sections. Several collections of round cells were seen in the fibrous tissue toward the edges of the section.

"The diagnosis is neurinoma of the seventh nerve within the middle ear."

The patient made an uneventful recovery with no postoperative fever or discomfort. The incision healed per primam intentionem, and the cavity and middle ear were epithelized and dry in two months. He was seen two years later in the

course of a routine check-up, and with the exception of persistent facial paralysis and deafness he appeared perfectly well. There was no local evidence of recurrence of the tumor, and the middle ear remained dry.

The case reported is unusual in several respects. Solitary neurinoma arising from the facial nerve in its canal is rare, only 12 cases having been found in the literature. Of these, in only 3 was the neurinoma reported as having arisen from the horizontal portion of the nerve in the middle ear.

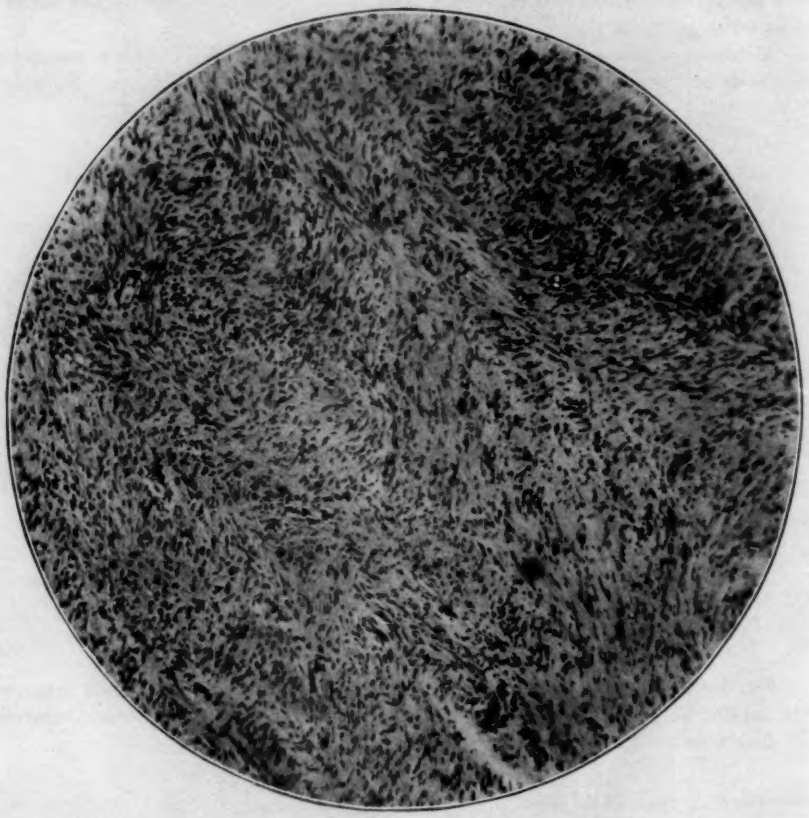

Fig. 5.—Low power photomicrograph showing the fibrous character of the tumor, with palisading and whorl formation of the nuclei.

The intervals between the onset of facial paralysis and aural symptoms and the discovery of the tumor are certainly the longest on record. The symptoms and the destructive process were minimal, considering their duration. This was undoubtedly due to the firm, fibrous, well encapsulated nature of the tumor. For the same reason it shelled out easily and removal was accomplished with a minimum of difficulty. Because of the fibrous nature and the extremely slow growth of the tumor, postoperative irradiation was not used.

SUMMARY AND CONCLUSIONS

Neurinoma of the facial nerve is a rare but definite clinical entity.

A brief survey of the history and pathology of neurinomas in general is presented.

Neurinomas are benign tumors producing symptoms by extension and pressure. In the case of neurinoma of the facial nerve, the first symptom is facial paralysis, followed after a period of years by deafness, chronic purulent otitis media and invasion of the mastoid.

Histologically, this tumor is characteristic of neurinomas, or neurofibromas, in general and presents a definite pathologic picture.

Only 12 cases have been found in the literature. These are reviewed, and a thirteenth case is reported.

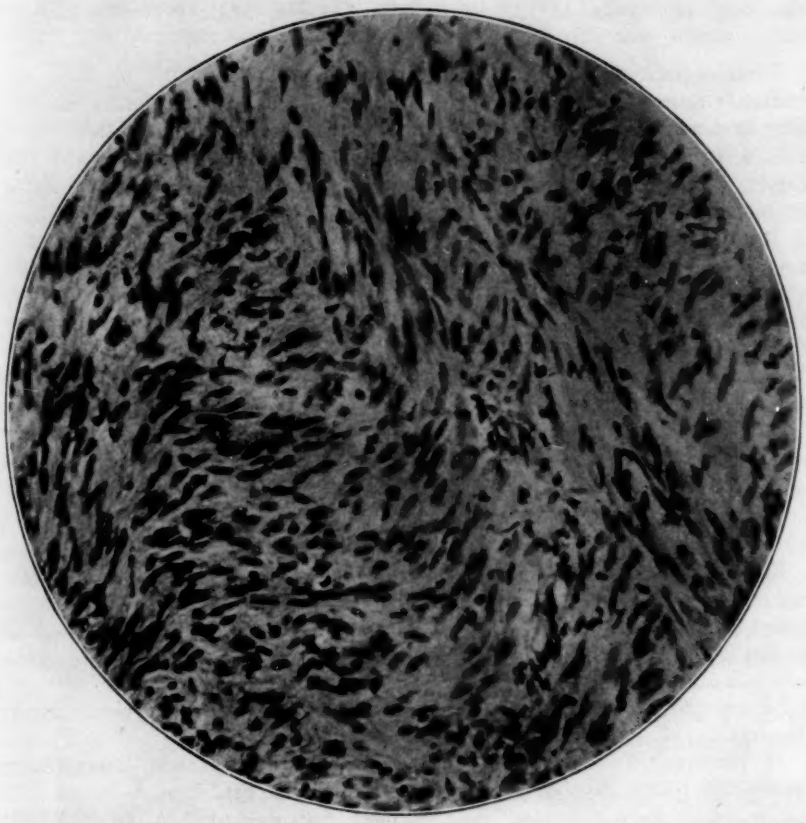

Fig. 6.—High power photomicrograph showing elongated, slender, curved nuclei arranged in eddies and fasciculi.

586 North Main Street.

Case Reports

BACILLUS PROTEUS SEPTICEMIA ACCOMPANYING ACUTE MASTOIDITIS WITH THROMBOSIS OF THE LATERAL SINUS

HARTWIG M. ADLER, M.D., AND CLAUDE KLAPPER, M.D., NEW ORLEANS.

That certain species of Bacillus proteus are pathogenic to man has gradually been proved. Infections with this organism frequently accompany urologic conditions and summer diarrhea of children. Such infections, always associated with other organisms, occur in other parts of the body. If looked for the bacillus would probably be observed more frequently in infections of the throat, nose and ear.

Reports in the literature are gradually accumulating to show that the cases of B. proteus septicemia associated with acute otitis media, acute mastoiditis and peritonsillar abscess, with consequent venous thrombosis and septicemia, carry a high mortality.[1] In 1935 Smith and Miller[2] reported a case of thrombosis of the lateral sinus caused by B. proteus. Positive cultures of material from the lateral sinus and the blood were obtained. The history was that of chronic otitis media complicated by thrombosis of the lateral sinus and septicemia. More recently McGovern,[3] Valle and Giroux,[4] Gerzog,[5] Neter and Chait[6] and others have reported cases of a similar nature.

REPORT OF A CASE

R. R., a man aged 28, was admitted on Nov. 5, 1941 to the contagious disease unit of this hospital with a condition diagnosed as malaria or typhoid fever. On rechecking the physical status on November 5, the intern found tenderness of the left mastoid, perforation of the left tympanic membrane and a foul-smelling

From the Department of Otolaryngology, Independent Service, Charity Hospital.

1. Kernan, J. D.: Two Cases of Infection by Bacillus Proteus, Laryngoscope **32**:304-310, 1922. Pagniez, P.; Plichet, A., and Decourt, P.: A propos d'un cas de septicémie à "Proteus," Bull. et mem. Soc. méd. d. hôp. de Paris **48**: 270-274, 1932.

2. Smith, A. B. P., and Miller, S.: Lateral Sinus Thrombosis Due to Bacillus Proteus, Lancet **1**:862 (April 13) 1935.

3. McGovern, F. H.: Fatal Otogenic Infection with Bacillus Proteus: Report of Two Cases, Arch. Otolaryng. **24**:618-621 (Nov.) 1936.

4. Valle, A., and Giroux, M.: Mastoïdite aiguë à Proteus vulgaris: septicémie, pleurésie, Laval méd. **2**:107-112, 1937.

5. Gerzog, B. C.: Bacillus Proteus in Otogenic Infection: Secondary Mastoiditis, Thrombosis of the Lateral Sinus and Bacteremia, Arch. Otolaryng. **30**: 275-279 (Aug.) 1939.

6. Neter, E., and Chait, R.: Bacillus Proteus in Otitis Media Complicated by Mastoiditis, Meningitis and Bacteremia, Arch. Otolaryng. **32**:947-950 (Nov.) 1940.

discharge from the left ear. Consultation with the otolaryngology staff was then requested.

History.—Two weeks before admission the patient had a recurrence of a discharge from the left ear, a periodic complaint for some eighteen years. He became ill six days later, was nauseated and vomited intermittently for three days. He was well for two days; then the vomiting recurred, and he had a severe chill. Two days later he had severe rigor, after which his temperature rose to 106 F. The next day he was brought to the hospital. At the time of his admission he had been unable to retain anything but fluids for two days. He complained of severe headache and some backache. He was irrational, and his sister, who furnished the history, stated that his behavior had been abnormal from the onset of the present illness.

Examination.—The patient was well nourished and well developed. He was in a semicomatose state and appeared acutely ill. His temperature was 102 F., his respiratory rate 25 and his pulse rate 80 per minute. Pronounced

Roentgenogram of mastoid, showing rupture through tegmen on the right.

tenderness was noted over the left mastoid region. The right pupil appeared larger than the left. The spleen was palpable. No other significant physical signs were present.

A roentgenogram, taken on November 7, showed chronic mastoiditis on the right side and acute mastoiditis on the left side, with rupture through the tegmen (figure).

Clinical Course.—On November 7, two days after admission, stiffness of the neck was noted. Lumbar puncture revealed a pressure of 300 mm. of water, 9 cells per cubic millimeter and a positive Queckenstedt sign on the left side. Blood cultures, taken on November 5 and 6, were reported to be positive for B. proteus.

Operation.—On November 8 mastoidectomy was performed on the left side. Exploration of a small perforation of the sinus plate revealed an extensive perisinal abscess. The sinus itself contained a well organized thrombus. The sinus wall was opened until free bleeding was obtained from above. No blood was obtained from below. The sinus was then collapsed above by placing a

pack between the wall of the sinus and the sinus plate. The left internal jugular vein, identified with difficulty because it was fibrosed, was sectioned between ligatures.

Course.—Despite the use of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole), sulfadiazine (2-[paraaminobenzenesulfonamido]-pyrimidine) and, finally, sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine), in addition to repeated blood transfusions, the postoperative course was steadily downhill. The patient continued to have a septic temperature and to vomit.

Nine days after operation he complained of a sharp pain in the right side of the chest, which was aggravated by breathing; a friction rub heard in the base of the right lung caused us to suspect the presence of a pulmonary infarct. A portable roentgenogram showed bronchopneumonic infiltration of the lower lobe of the right lung. A second roentgenogram, taken ten days later, showed extension of the process. On November 27, the nineteenth postoperative day, the abdomen became greatly distended, and the skin took on a yellow hue. The patient died on December 1.

Complete Laboratory Data.—The results of laboratory examinations are tabulated as follows:

Date	Hemoglobin, Gm. per 100 Cc.	Blood Red Cells	Blood White Cells	Polymorphonuclears, %
11/ 5/41	13.8	5,700,000	10,000	72
11/ 6	....	4,500,000	12,900	92
11/13	....	4,000,000	13,400	..
11/15 and 16	Blood smear showed no malaria plasmodia			
11/20	....	3,200,000	11,400	92
11/26	....	4,600,000	33,600	90

Urine

11/ 5 Essentially normal; cultures negative for Bacillus typhosus, Bacillus paratyphosus and Bacillus dysenteriae

11/28

11/28 Essentially normal

11/29 Essentially normal

Blood Chemistry

11/ 6 Urea nitrogen 16.1 mg. per 100 cc.

11/15 Carbon dioxide-combining power 40 vol. per cent

Serologic Reactions

Wassermann, Kline and Kolmer reactions of the blood negative

Spinal Fluid

11/ 7 Initial pressure 300 mm. of water; positive Queckenstedt sign on left; fluid clear, with 9 cells per cu. mm.

11/ 8 Pressure 335 mm.; Queckenstedt reaction positive on left; fluid clear, with 17 cells, predominantly lymphocytes; trace of globulin; culture sterile

11/11 Pressure 250 mm. of water; positive Queckenstedt reaction on left; fluid clear, with 3 cells per cu. mm.; no globulin; sugar 69 mg. per 100 cc.

11/28 Pressure 210 mm. of water; fluid clear, with less than 10 cells per cu. mm.; no globulin; culture sterile

Bacteriologic Data

11/ 5 Blood culture yielded 1 colony of B. proteus per cc.; agglutination test positive for B. dysenteriae Flexner type in 1:320 dilution

11/ 6 Blood culture yielded innumerable colonies of B. proteus per cc.

11/ 7 Smear of discharge from right ear positive for Shigella dysenteriae

11/ 8 Blood culture negative for B. proteus

11/ 9 Pus from mastoid at operation positive for Escherichia coli

11/17 Blood culture negative

11/23 Blood culture negative

Clinical Diagnosis.—The clinical diagnosis was acute mastoiditis with thrombosis of the lateral sinus, B. proteus septicemia and pneumonitis.

Autopsy.—Autopsy revealed acute mastoiditis with thrombosis of the lateral sinus; extradural abscess; bilateral pulmonary abscess, resulting from pneumonia and empyema; congestion of the spleen, and focal necrosis of the liver.

COMMENT

We believe that the perisinal and extradural abscess, present at the time of operation and not completely drained, must, through collateral venous circulation, have given rise to the septic emboli responsible for the infarct in the right lung, to which the patient's death can reasonably be attributed.

It is our impression that the newer chemotherapeutic drugs might have been effective had the abscess been more adequately drained.

Charity Hospital.

Clinical Notes; New Instruments and Technics

METHOD OF ROTATING THE OPEN SAFETY PIN IN THE ESOPHAGUS

PAUL G. BUNKER, M.D., ABERDEEN, S. D.

The removal of the open safety pin in the esophagus, point up, has long presented one of the most interesting problems in the field of endoscopy. Many ingenious means of doing this have been recommended. I wish to present a method which has proved successful and easy to accomplish, both in my limited series of 7 patients, ranging in age from 4 months to 24 years, and in experiments on dogs.

The present method has the following advantages:

1. The forceps can be applied easily. This procedure, in accordance with the usual methods (dangling, sheathed point, ring application, etc.), has always been a difficult thing for me to do, especially when it is necessary to work through a 4 or 5 mm. esophagoscope. The forceps occupies so much of the lumen in the smaller esophagoscopes that it is difficult to visualize the foreign body. By the method here presented it is necessary only to bring the distal end of the esophagoscope into the proper relation to the keeper end of the pin, and the forceps can easily be applied blindly. To do this, the blades of the forceps are spread inside the esophagoscope as it is inserted so as to impinge against the inner wall. It can then be felt to slide out the distal end of the esophagoscope, and it is now necessary only to overbite the forceps the estimated distance to engage the pin properly. The instrument used for this purpose is the ordinary flat, forward-grasping forceps (Chevalier Jackson model) with the serrated grasping surface (figs. 1 to 3).

2. It is a one stage procedure. That is, the pin can be rotated and withdrawn in one manipulation. In the event the forceps should slip off, it can easily be reapplied. If this happens, it will be after the pin has been rotated (see report of case). The ring end of the pin can then be grasped and the pin removed without difficulty.

3. This method at no stage violates the principle of the after-trailing point, so that there is no danger of perforation.

4. It is not necessary to go down after the point or the ring of the pin. Figure 1 *1* shows the usual position of the safety pin in the esophagus. The keeper shaft of the pin lies nearly parallel to the esophageal wall, while the pointed end lies at an angle to it and below the keeper. It is this fact which makes my method applicable. Any attempt to pass the esophagoscope down far enough to grasp the point or the ring necessitates by-passing the keeper end. This is likely to dislodge the pin, causing it to disappear into the stomach, as happened in 1 of my cases.

5. No special instruments are required.

METHOD

The principle here involved is the making use of the beveled end of the esophagoscope, both for the application of the forceps and for the rotation of the pin. The esophagoscope is introduced in the usual manner until the keeper of the pin is sighted. Then the esophagoscope is rotated through an arc of 90 degrees (fig. 1 *2 A*), so that the keeper fills the entire field of the esophagoscope. At this point, if necessary, the keeper can be displaced toward the midline of

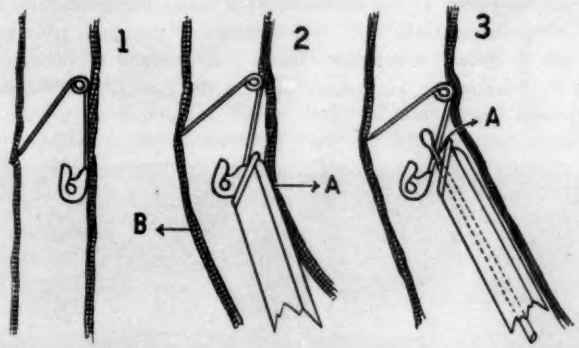

Fig. 1.—*1,* usual position of open safety pin in esophagus; *2,* bringing of pin into proper position for this method; *3,* application of forceps.

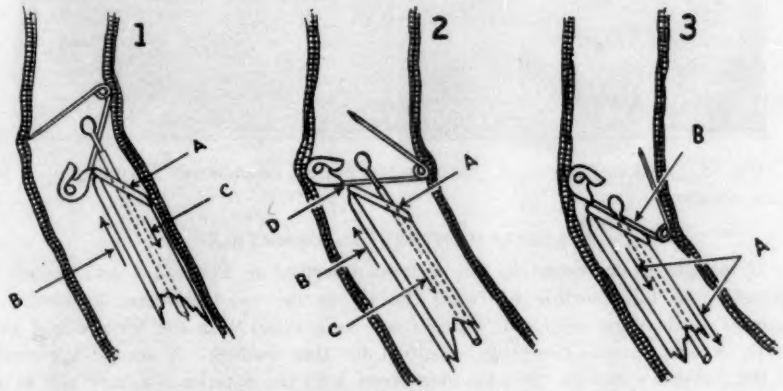

Fig. 2.—*1,* rotation of esophagoscope around forceps; *2,* rotation of pin around tip of esophagoscope through counterpressure with tip of esophagoscope and traction with forceps; *3,* position of pin after rotation.

the esophagus by slightly adducting the head and neck (fig. 1 *2 B*) toward the shoulder of the keeper side; the tip of the esophagoscope, used as a gentle lever (fig. 1 *2*), then brings the pin into the proper position. The forceps is now applied in the manner previously described (fig. 1 *3*). Application of the forceps at any point between the keeper and the ring of the pin is satisfactory for carrying out the manipulation. It is important to overbite the keeper shaft, so that the pin is engaged between the shanks of the forceps, and not between the

grasping surfaces (fig. 1 *3 A*). This allows the pin to rotate freely within the shanks. The grasping surface serves merely as a guard to keep the forceps from slipping off the pin.

The esophagoscope is now rotated through an arc of 180 degrees, which reverses the relative position of the beveled end of the esophagoscope (fig. 2 *1 A* and *2 A*). Downward pressure is now brought to bear on the keeper of the pin (fig. 2 *1 B* and *2 B*) with the tip of the esophagoscope, while traction is made with the forceps (fig. 2 *1 C* and *2 C*). This rotates the pin right around the beveled end of the esophagoscope, as shown in figure 2 *2 D*. Figure 2 *3* shows the position of the pin after rotation, in which position the pin, esophagoscope and forceps can now be safely withdrawn (fig. 2 *3 A*) without reapplying the forceps. This is done by holding the pin snugly against the beveled end of the esophagus as it is withdrawn (fig. 2 *3 B*).

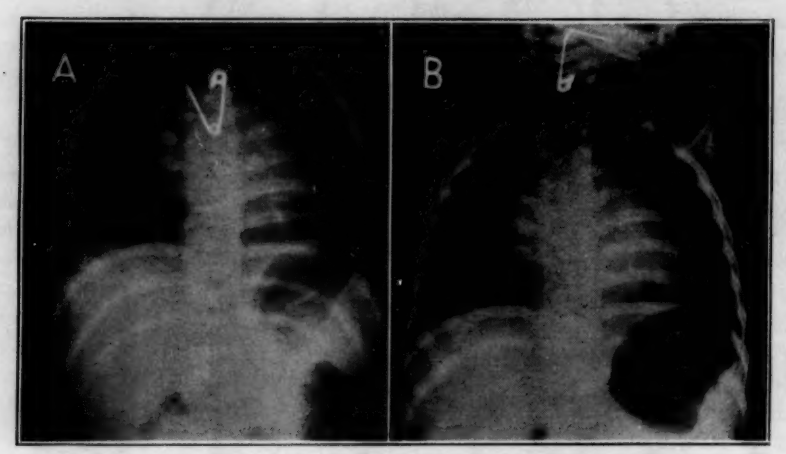

Fig. 3 (present case).—*A*, open safety pin in esophagus; *B*, position of pin after rotation.

SUPPLEMENTARY INFORMATION

If the pin is not found in the position described in figure 1 *1* and cannot be brought into this position by using the tip of the esophagoscope, as shown in figure 1 *2*, then the keeper of the pin can be grasped with the forceps and very gently drawn up into the proper position for this method. A second application of the forceps would be required to proceed with the rotation. I have not as yet encountered a case in which this was necessary in a human patient but have found it easy to do on a dog, and I see no reason that it could not be done on the human patient.

Theoretically, it seems logical to have a notch placed in the tip of the esophagoscope to prevent the pin from slipping off the end of the esophagoscope as the pin is rotated. Practically, I have had no difficulty with the pin's doing this and therefore have not used this device, though it could easily be incorporated into the technic.

If the pin is lodged in the cricopharyngeal region, it may be more easily rotated, after the proper application of the forceps has been made, by lowering the pin into the thoracic portion of the esophagus, where there is more room to carry out this manipulation.

REPORT OF A CASE

Baby D. N. Jr., aged 7 months, was referred to me on May 4, 1940, by Dr. J. O. F. Kraushaar for removal of an open safety pin from the esophagus. Figure 3 *A* shows the position of the pin before removal and *B* its position after rotation. After rotation of the pin, the forceps slipped off as the foreign body and esophagoscope (5 mm.) were being withdrawn. This is the only case of mine in which this has happened. When the esophagoscope was reintroduced the baby's respirations immediately ceased. The second roentgenogram (fig. 3 *B*) was then taken to determine the cause of this and to relocate the pin. This exposure showed the pin in the hypopharynx, rotated, with the keeper in the larynx. A laryngoscope was then introduced and the pin removed without difficulty. The baby made an uneventful recovery.

This case is presented because the necessity of taking the second roentgenogram made it possible to show definitely that a safety pin can be safely rotated by this method, even in a very small infant, when a small esophagoscope is essential.

SUMMARY

A new method of rotating an open safety pin within the esophagus is presented which can be carried out simply and with safety to the patient.

Prof. E. K. Baillie, Fine Arts Department of Northern States Teachers College, made the diagrams used in this paper.

Newberry Building.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

FUNCTIONAL EXAMINATION OF HEARING

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AND
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As has been stated in our reviews of the past few years, reports on the functional tests of hearing, especially those involving tuning forks, are few. Audiometry receives a fair amount of attention. The greater part of this review deals with the conservation of hearing and the effect of war on hearing.

Various military requirements, recommendations and methods of testing and the effects of noise and trauma on hearing are reported.

Much foreign literature has not been accessible; that available has been abstracted.

A practical method for calculating the percentage loss of hearing for speech from the audiogram has been formulated. The method as evolved by the Council on Physical Therapy of the American Medical Association and its consultants and accepted by the House of Delegates of the Association is reported.

HEARING TESTS

Bunch [1] described the development of the audiometer. First called *Hörmesser* by Politzer in 1877, it has been variously termed acumeter, audimeter and sonometer. The Council on Physical Therapy of the American Medical Association gives the following definition: "A clinical audiometer is an instrument for measuring the acuity and range of hearing."

Credit for the earliest application of the electric current to this purpose lies between Hartmann and Hughes, in 1877 and 1878 respectively. The work of Hartmann did not receive much attention, but Hughes's instrument was described in textbooks as late as 1893. Instruments credited to de Lacharrière, Cozzolini, Cheval, Jacobsen,

1. Bunch, C. C.: History of the Development of the Audiometer, Laryngoscope **51**:1100 (Dec.) 1941.

Seashore, Tretop, Gradenigo, Foy and Stefanini are mentioned. Seashore's audiometer was used in the Chicago public school survey.

Bezold's "continuous tone series" of tuning forks is referred to, and the audiometer developed by Dean and Bunch, which gives a relatively pure tone throughout the entire hearing range, with controllable and measurable intensity, is described. This instrument had a tone interrupter and a signal light and key. It was the first of the modern type of instruments.

The next step was the application of the vacuum tube to radiotelephony by Minton and Wilson, followed by the modern Western Electric 1A audiometer, presented to the medical profession by Fowler and Wegel for testing the threshold of audibility at various levels. The Kranz model and the Knudsen-Jones Sonotone, the Maico D-5 audiometer and the Western Electric 6B type are accepted by the Council on Physical Therapy of the American Medical Association (as of May 1941).

The greater part of the present day studies in audiometry originated in this country, but reports from England and Sweden (Bárány; Holmgren) have appeared recently.

Trowbridge [2] presents the advantages of audiometric testing of hearing, which he calls electroaudiography, and gives an analysis and interpretation of the audiogram. His presentation is more or less a résumé of the interpretations of otologists in the field of audiometry. He summarizes the chief uses of the electroaudiogram as follows: to detect and differentiate acoustic deficiencies, to discover obscure perceptive defects, to measure functional response, to estimate the value of therapeutic effects, to observe and record the progress of the hearing conditions, to provide documentary data for medicolegal cases and to prescribe hearing aids. He emphasizes the importance of detecting acoustic deficiencies long before serviceable hearing is impaired. This is necessary if restoration to normal is to be obtained. In methods other than electroaudiography factors are too variable to insure reliable results and to make possible the early diagnosis of incipient impairment of hearing. Trowbridge also shows how the characteristic tracings and audiographic patterns aid in classifying a hearing defect and in determining a possible etiologic background. The conductive deficiencies show defects in the 128 to 1024 cycle range, rarely exceeding a loss of 20 to 30 decibels, with usually no abrupt or pronounced deflections from the isoelectric line. Perceptive impairments show losses for frequencies above 1024 cycles, and when they involve the ranges below 1024 cycles the loss usually exceeds 30 decibels. With the mixed type

2. Trowbridge, B. C.: Electroaudiography: Analysis and Interpretation of the Audiogram, Arch. Otolaryng. **35**:899 (June) 1942.

of deafness the loss usually exceeds 25 to 30 decibels throughout the entire range. Trowbridge also points out the audiographic pattern observed in children with deafness due to obstruction of the eustachian tubes by excessive lymphoid tissue. This pattern shows losses in the perceptive range with little change in the conductive range or the lower frequencies. This type of deafness is not due necessarily to a lesion of the inner ear or nerve. He states that focal and systemic sources of infection are responsible for the majority of perceptive defects. The hearing loss occurs in the upper limits of and above the range of serviceable hearing. The pattern can also give one information concerning the cause of the impairment in hearing, the activity of the pathologic process and the duration of the acoustic lesion. In cases of early impairment the loss is abrupt and progressively irregular. At times defects take on a stair step pattern, in which successive frequencies show the same amount of loss. These irregular defects are associated with a gradual and progressive depression for the higher frequencies in the later stage of acoustic impairment. Local infections of long duration may be associated with conductive defects, in addition to the perceptive changes in the audiogram. Syphilis may produce perceptive defects similar to those associated with focal infection. In presbycusis the hearing for frequencies below 1024 cycles remains practically normal up to 70 years of age. At 50 to 60 years of age there is usually some involvement of hearing for frequencies above 2048 cycles, with the higher ranges showing the greater loss. Thus, gradually the lower and the middle portion of the scale are affected if the impairment continues. Abrupt and irregular losses in the audiogram are rarely observed. In otosclerosis there is usually the same amount of loss for the higher as for the lower frequencies in the early stages. Later there is greater loss for the high tones.

Trowbridge divides traumatic deafness into three types: occupational, detonation and aerovibration. The occupational type first affects the middle and later the upper limit of the auditory range. It is usually bilateral, following the same general pattern in both ears, but is usually worse in one ear. In detonation deafness, due to a sudden loud noise near the ear, the defect is usually more pronounced in one ear. Impairment is usually observed at the high frequencies, at and above 4096 cycles, but may occur at all frequencies. In aerovibration deafness the defect early is at the 4096 cycle frequency, then spreads fanwise and with continued exposure eventually involves the speech range.

Electroaudiography not only measures the threshold of hearing but enables one to analyze the entire area of audibility with considerable precision. By this means word deafness, in which higher brain centers are involved, can be differentiated from physiologic deafness. With

word deafness little change is found for pure tone hearing as shown by the audiogram. Audiography also aids in making a prognosis. Whenever there is a total loss for any one of the frequencies, or when the average loss for the speech range exceeds 30 decibels, one should make a guarded prognosis. The author also points out the importance of audiograms in the evaluation of therapeutic measures and in the fitting of hearing aids. He feels that the information obtained from the audiometric examination is so important that the otologist himself should make the test. This procedure has standardized the diagnosis, simplified the prognosis and rationalized the therapy of defective hearing.

No mention is made of bone conduction determinations and their value in making an accurate diagnosis.

Connell and Trowbridge [3] reiterate the statements made in another article.[2] They emphasize the importance of a careful history and examination, the use of tuning forks, particularly for the Weber, Rinne and Schwabach tests, to determine the type of deafness and audiometric tests for pure tones, speech and bone conduction. They state that tuning fork tests alone cannot give an intelligent analysis of the hearing condition. They point out the value of early detection of hearing defects. Conductive defects in general are amenable to local therapy of the ears, nose and throat, and perceptive defects require a thorough systemic investigation to locate the etiologic agent before therapy can be instituted. Response of the hearing mechanism to therapy is usually slow and requires diligent and patient supervision by the otologist for a favorable outcome. Periodic aural health examinations, even after school age, seem to be as justified as periodic general health examinations.

Wishart [4] discusses the mistakes frequently made in the diagnosis and estimation of deafness. He does not believe that tuning forks are at all necessary for a complete hearing test. Few otologists have tuning forks of the low frequencies, such as 32 to 64 double vibrations, or means for testing above 4096 double vibrations unless they use whistles and the monochord. If the latter are used, the actual pitch and intensity are not always reproduced with certainty. The Weber test is not essential, and the Rinne test can be made much more quickly and accurately with the audiometer. This is done by obtaining both the bone and the air conduction threshold for 512 double vibrations. If the difference between these thresholds is more than 45 decibels, the Rinne reaction will be positive; if it is less than 45 decibels, the Rinne reaction will be negative. The two common factors of error in the standard Rinne test made with the tuning fork, namely, the error due to the constant decrement of the

3. Connell, E. S., and Trowbridge, B. C.: Essential Procedures in the Diagnosis of the Abnormalities of Hearing, with Some Audiometric Findings, Laryngoscope **52**:545 (July) 1942.

4. Wishart, D. E. S.: Mistakes Made in the Diagnosis and Estimation of Deafness, J. Michigan M. Soc. **41**:861 (Oct.) 1942.

fork and the error due to possible variations in pressure and in the selection of the point of contact, are eliminated with use of the audiogram.

Certain precautions, however, must also be taken with the employment of the audiometer. First of all, a soundproof room should be used for testing. The opposite ear must be properly masked. Wishart suggests the use of a tightly packed cotton ear plug impregnated with a solution of wax and paraffin over which a large ear pad of soft sponge rubber is placed. In the center of the pad is the telephone ear piece which causes the masking sound. The masking sound is provided by an electric buzzer, the intensity of which can be varied. Sound from the opposite ear under most circumstances will reach the masked ear if hearing in the masked ear is 40 decibels greater than that in the tested ear. Therefore the estimation of hearing in a seriously impaired ear will be imperfect. The proper fitting of the ear piece to the ear is essential, and Wishart describes a method for doing so by strapping the ear piece to the ear snugly by means of a plain strap of webbing placed around the ear.

Errors with the audiometer are due to failure to have the machine properly calibrated and adjusted. This must be constantly watched for. Improper technic in testing leads to faulty results. The author suggests that each pitch be tested for by approaching first from above threshold and later from below. Results should be checked. The patient's intelligence and type of response must be taken into consideration. Also, the difficulty in differentiating between a real fading sensation and a lingering memory image must be considered. Introduction of adventitious sounds from the machine itself may occur and must be carefully watched for. Wishart stresses the fact that there is no short cut to or easy way of making a diagnosis of deafness. The examiner should do his own testing, under proper conditions. A complete history and examination, including a nasopharyngoscopic study, are essential. Analysis of the curves and their interpretation require experience and may be difficult, especially when one is attempting to determine the percentage loss of hearing. Many of the curves supplied by the makers of audiometers which show a complete loss of serviceable hearing are inaccurate. It should also be remembered that audiometric charts for a patient cannot be correlated with those taken elsewhere with a different machine. An audiometer record can be a great source of satisfaction when taken with every attempt to avoid error. Otherwise such a record means nothing to the examiner or to any one else who may get it.

Huizing and Pothoven [5] compare the advantages and disadvantages of the various procedures for testing hearing, which they classify as

5. Huizing, H. C., and Pothoven, W. J.: Classical and Modern Methods in the Evolution of Functional Hearing Tests, Ztschr. f. Hals-, Nasen- u. Ohrenh. **47**:390, 1941.

follows: (*a*) hearing tests for speech; (*b*) Hartmann fork tests; (*c*) measurement of the half-time duration for forks (Royal Society of Medicine); (*d*) measurement of the duration of tone (Struycken), and (*e*) audiometric measurements.

(*a*) Tests of acuity of hearing for speech may be made more uniform by using a phonograph and a controllable amplifier.

(*b*) The decrement must be calibrated, and the initial energy must be uniform.

(*c-d*) The forks are standardized and calibrated and timed to one-half their duration. The number of seconds the sound is heard is compared with the normal; each second lost equals 6 decibels. The decrement is affected by the manner of holding the fork. The Bezold forks are considered reliable. They are activated by a rubber-covered hammer. The authors have constructed a table converting seconds of duration into decibels of hearing loss. In a room with 25 decibels of noise this method is not reliable for hearing losses of less than 40 decibels. The authors have also constructed a table for the Struycken forks. The initial intensity for 250 cycles is 48 decibels, and that for 500 and 100 cycles is 56 decibels. Therefore these forks are usable only in cases of mild deafness. The higher forks have a 65 decibel intensity, but their duration is only fourteen seconds.

The authors cite the United States Public Health Service survey,[6] which divides deafness into four grades: (1) inability to hear in public lecture halls, etc., average loss 25 decibels; (2) inability to hear normal speech at 1 meter, average loss 45 decibels; (3) inability to hear telephone conversation, average loss 65 decibels, and (4) inability to conduct conversation under any condition, average loss 85 decibels.

(*e*) The authors state that the differential diagnosis of middle and inner ear deafness cannot be made on the basis of fork tests alone; therefore it is to be expected that audiometric and fork tests will not have comparable results. They cite the recruitment phenomenon, which they credit to Fletcher and Munson and which is not demonstrable by tuning forks, as one reason for the varying results.

The audiometer is an instrument of precision, the proper use of which requires the joint knowledge of the otologist and the acoustic physicist. Even in a so-called quiet room there is some masking effect, especially for low tones. When there is a wide difference in the hearing of the two ears, the carrying over of the sound to the good ear must be considered. The problem of masking is not yet settled. A more detailed analysis of the masking sound is necessary.

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6. Preliminary Analysis of Audiometric Data in Relation to Clinical History of Impaired Hearing, Bulletin 2, The National Health Survey 1935-1936, Hearing Study Series, United States Treasury Department, Public Health Service, 1938.

Goalwin [7] presents a method for the use of tuning forks in audiometric tests when an audiometer is not available. The rate of decay for each fork must first be measured. This is determined by comparing the results of tuning fork tests with the examiner's own audiogram for both air and bone conduction. The difference in decibels for each pitch is then recorded in a table. Tuning forks for each pitch are then tested for both air and bone conduction, a stopwatch being used to obtain the difference in vibration time between air and bone conduction for both the audiogram and the tuning fork. The figure representing the difference expressed in decibels for air and bone conduction as determined by the audiometer is divided by the difference expressed in seconds for air and bone conduction as determined by the tuning fork of the same pitch. The result is the rate of decay of that tuning fork in decibels per second. In testing a patient, the difference in seconds between the patient's and the observer's hearing is determined for a tuning fork of each pitch. This figure is divided by the time required for 1 decibel of decay for that pitch; the resulting time obtained is the hearing deficit of the patient for that pitch. To plot the patient's audiogram, a standard audiometer chart is used on which the observer's audiogram has been charted. The observer's air and bone conduction lines are taken as normal. The tuning forks should be held and applied in the same manner for every test. The patient's hearing loss for a given pitch is determined by multiplying the hearing deficit for that pitch expressed in seconds by the rate of decay of the fork used expressed in decibels per second. This hearing loss is relative to the observer's own hearing. To obtain the absolute hearing loss one makes allowances for the observer's own acuity of hearing. The best tuning forks to be used for this purpose are those having a rate of decay of about 1 decibel per second. When new tuning forks are to be purchased, those with an air-bone differential of thirty to fifty seconds are the best. A quiet room is essential for testing. Goalwin states that this method of testing is not the procedure of choice, but one of necessity.

We suggest that rather than make allowances for the observer's own hearing, the mean average in seconds for vibrating time for both air and bone conduction could be obtained for each fork by testing a group of persons with normal hearing. If this value were used in computing the results, a more accurate actual absolute hearing loss for the patient would be obtained. In addition, for more accurate and standard results the rate of decrement for each fork used could be first determined by the Bureau of Standards, Washington, D. C.

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7. Goalwin, H. A.: The Use of Tuning Forks for Audiometry, M. Bull. Vet. Admin. **18**:266 (Jan.) 1942.

Senturia and Thea [8] review the literature concerned with observations on bone conduction in audiometry. They also report some preliminary observations of their own. Three audiometers were used, the Western Electric 1A, the Western Electric 6B and the Maico D-5. The tests were performed in a soundproof room. Bone conduction was tested with the receiver placed over the mastoid process. Masking of the opposite ear was obtained by using either a battery set producing a complex noise of 60 decibels or the masker on the Maico instrument. Twelve normal persons were tested to obtain normal curves. Each of the machines gave a different curve. The Maico D-5 audiometer curve always showed a slight rise at both ends, with the center tones forming a flat base. The Western Electric 1A instrument revealed a progressive rise in the upper ranges, with flattening in the lower portion of the curve. The Western Electric 6B audiometer curve formed an irregular V, the left arm rising at a slightly sharper angle than the right. The great variation in the curve of the Western Electric 1A audiometer may be due to the greater bulk and weight of the bone conductor of that machine. The Western Electric 1A instrument permitted testing of the entire range; the other two were more limited. No tests were made for frequencies below 256 double vibrations because the vibrations were felt. Tests for 8192 double vibrations could be obtained readily only with the Western Electric 1 A and 6 B instruments.

Three types of patients were tested: those with nerve lesions; those with conductive lesions, including otosclerosis, and postoperative patients. The patients with nerve, or perceptive, lesions showed an average loss of 15 to 35 decibels from the normal; the bone conduction curve followed the air conduction curve, diverging more in the higher frequencies. The most frequent and greatest loss occurred at 2048 and 4096 double vibrations. In a large number of patients hearing for the lower tones remained within normal limits. Syphilitic patients revealed almost complete loss, with only moderate reduction for the middle and high tones. The patients with bone conduction defects gave repeatedly excellent bone conduction responses. The authors conclude that in view of the great discrepancy among the bone conduction curves obtained with various makes of audiometers, a normal curve must be made with each instrument and checked frequently. Bone conduction tests for a single frequency may give erroneous information. Bone conduction curves should be for a complete range of frequencies if possible, or at least for 512, 2048 and 4096 double vibrations. With increasing age there is a decrease for bone conduction in the high tones similar to that for air conduction. Persons with otosclerosis show only a slight deviation from the normal

8. Senturia, B. H., and Thea, A. R.: Bone Conduction in Audiometry, Laryngoscope **52**:675 (Sept.) 1942.

bone conduction curves. Nasal obstruction, allergic rhinitis and tympanic perforations have little effect on bone conduction. The region of 4096 double vibrations appears to be the most vulnerable portion of the bone conduction curve, and the range below 1024 double vibrations, the most resistant. Like all other tests, determinations of bone conduction should be made in a soundproof room.

Weersma,[9] who investigated the influence of noise on the hearing of normal and of deafened persons, cites the pioneer work of Wegel and Lane, who found that the masking effect is greatest when the masking tone is of approximately the same frequency as the one to be masked; if the masking tone is higher the disturbance is slight; if the masking tone is lower there is at first little effect, but with greater intensity a strong effect is noticed. That is, the masking effect is dependent on both pitch and intensity. Weersma used an amplifying system ending in a funnel-shaped loud speaker, which was placed above the head of the person to be examined. For masking noises various devices were used: these included an electric buzzer; a "thermic noise maker," which operated by amplifying the brownian electronic movements and covered a continuous sound spectrum; a system of filters whereby this spectrum could be utilized in any part, and a measurable intensity control. What corresponded to a pure tone audiometer was used for the tones to be masked and measured. The author reminds that the ear perceives tones of from 800 to 5000 cycles at a much lower intensity than tones of other frequencies, as shown by Fletcher and Munson and others, and he presents a series of graphs illustrating his results.

In normal persons the masking effect of the thermic noise at 40 decibels began at 100 to 200 cycles and increased up to 800 cycles. If the intensity of the noise increased to 86 decibels, the effect was greater up to 600 cycles and the rise more sudden. When the character of the noise was changed by filtering the spectrum, the effect became more uniform across the graph. The graph for the buzzer resembled that for the thermic noise without filtering.

It was found that persons with deafness for low tones were less disturbed by the masking than were normal subjects. On the other hand, patients with deafness for high tones were greatly affected. From the practical standpoint, outside noise is largely composed of low tones, which mask the better heard sounds for persons with deafness for high tone and make hearing more difficult for them. The graph illustrates what happens in cases of presbyacusis.

Mueller[10] describes the procedure used at the Massachusetts Eye and Ear Infirmary under the Winthrop Foundation for the Study of

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9. Weersma, P.: The Influence of Noise on the Tone Hearing of Normal and Poor Hearing, Ztschr. f. Hals-, Nasen- u. Ohrenh. **47**:402, 1941.

10. Mueller, W.: The Clinical Examination of the Hard of Hearing, Ann. Otol., Rhin. & Laryng. **51**:756 (Sept.) 1942.

Deafness for examination of hard of hearing patients whose hearing disabilities could not readily be ascribed to an obvious pathologic condition of the ear. A complete history is first taken, and careful examination of the external ear and tympanic membrane is made. The patency of the eustachian tubes is determined. The nose, sinuses, nasopharynx, pharynx and larynx are then carefully examined. The actual hearing tests consist of the Gelle test; determinations with the pure tone audiometer for both air and bone conduction, and tests for words with the pure tone audiometer with phonograph attachment. The last test is used without amplification to determine the percentage of words actually understood by the patient, and then with amplification sufficient to raise the score to well over 90 per cent. This method has been a good check on the pure tone audiograms. The Weber test is carried out with a fork of 256 or 512 cycles per second. The Rinne test is performed with a fork of 512 cycles.

The prekindergarten group, namely, children under 3½ years of age, were tested by the so-called play conditioning technic. This involves conditioning the patient to the sound of bells, rattles, whistles, etc. The responses are obtained by having the child activate various toys.

De Crinis [11] describes a case of deafness for speech with retention of hearing for pure tones following a cerebral accident. He explains that a lesion above the nucleus of the cochlear nerve affecting only the crossed fibers may cause such a syndrome. The lesion may also be located at the medial geniculate body or the two posterior corpora quadrigemina.

The symptom complex described by Guttich, i. e., loss of hearing for speech with retention of good hearing for tones, associated with unilateral loss of caloric reaction, is explained by such a lesion of the midbrain, which may be of encephalitic origin and may involve the posterior longitudinal bundle.

Lipman and Grassi [12] measured the auditory thresholds of 18 young men and 11 dogs at seven octave frequencies from 125 to 8000 double vibrations under comparable physical conditions. They found that man and the dog hear equally well at the lower frequencies, but that the dog has a distinct superiority in the higher ranges.

Novotny [13] describes a case of osteoma of the external canal with internal ear deafness. In 4 similar cases previously cited from the literature only conduction deafness was present. The author believes it possible that inflammatory reactions due to the closure of the canal may have caused damage to the cochlea by toxemia.

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11. de Crinis, M.: Speech Deafness with Retained Hearing for Sound, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **149**:183, 1941.

12. Lipman, E. A., and Grassi, J. R.: Comparative Auditory Sensitivity of Man and Dog, Am. J. Psychol. **55**:84 (Jan.) 1942.

13. Novotny, O.: Osteoma of the External Canal Causing Deafness, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **149**:5, 1941.

Hughson and Thompson [14] present a method for determination of the acuity of hearing in relation to speech reception, or the ability to understand speech. A normal level is established, and then values for a series of groups of persons presenting varying degrees of impairment are contrasted with it. The equipment for this test consists of a microphone, an amplifier with a built-in volume indicator, a General Electric radio T pad attenuator and a loud speaker. The microphone and amplifier are located in a soundproof booth. This is connected by a 500 ohm line to the attenuator, which, with the loud speaker, is in an adjoining soundproof room. A transformer couples the attenuator either to a telephone receiver or to the voice coil of the speaker. A diagram of the equipment and setup used accompanies the article. The test is performed by a reader, who recites a given list of sentences into the microphone, situated in the small soundproof booth. The reader learns to speak at a uniform loudness level, aided by the visual guidance of the volume indicator incorporated in the amplifying system of the apparatus. Men and women readers can be interchanged arbitrarily. Identical response levels were obtained regardless of the quality of the reader's voice. This method is preferred to the phonograph records, as the spoken voice permits of much greater flexibility in carrying out the test. The sentences are properly spaced, which gives the subject ample time to repeat the words. Either ear phones can be used to test monaural hearing, or the loud speaker, to determine binaural hearing. The subject is placed at a fixed distance, 10 feet (3 meters), from the loud speaker. The operator controls the loudness of the spoken words delivered by means of the attenuator. The level at which the subject first hears the reader's voice is taken as the threshold of audibility for speech. It is then gradually decreased until the subject can no longer repeat at least two thirds of the sentences correctly.

The normal threshold level was found to be 67.3 decibels of attenuation of the maximum intensity of the acoustic system. This is comparable to the zero level of the standard audiogram. A series of groups of persons having varying degrees of deafness were tested in a similar manner, and a definite correlation between the audiometric percentage loss and the percentage loss for speech was determined and presented in the form of a chart. The average audiometric percentage loss was computed by averaging the values for the frequencies 512, 1024 and 2048 and multiplying by 0.8. The percentage loss for speech was arrived at by determining the observed departure from the established normal in terms of direct arithmetical calculation, the normal attenuator setting of

14. Hughson, W., and Thompson, E.: Correlation of Hearing Acuity for Speech with Discrete Frequency Audiograms, Arch. Otolaryng. **36**:526 (Oct.) 1942.

67.3 decibels being regarded as the zero threshold level. The authors show a constant relationship throughout the entire audiometric range of intensities between the percentage loss of total hearing and the percentage loss for speech, although they are unequal in degree. Bone conduction and nerve deafness as such are not significant factors in the percentage impairment of speech reception thresholds. Frequencies above and below 512 to 2048 have little significance in the subject's ability to understand speech. Hughson and Thompson state that this test accomplishes two main objectives, first, simplicity of performance and appraisal of observations, and, second, a direct correlation of the audiogram with some standard speech reception test.

Sabine [15] suggests a means of making a quantitative evaluation of useful hearing based on experimental data. This should be useful to otologists, who are frequently asked to testify as to what percentage loss of useful hearing has resulted from injury. Sabine explains in detail how he developed his chart, the work done by Bunch, the United States Public Health Service, Knudsen, the Bell Telephone Laboratories, Fletcher and Steinberg and Gardner being taken into consideration. This chart gives the percentage loss of useful hearing for the speech range. By applying the audiogram of the patient to the chart the percentage loss of useful hearing for that patient can be figured. To compute the total percentage loss of hearing for both ears, the percentage loss for each ear is first determined and then one eighth of the percentage loss for the poorer ear is added to that for the better ear. The sum of these values will be the total percentage loss of hearing for the patient. A complete understanding of this chart and the method employed will require a careful perusal of this paper.

In the discussion on this paper, the chart and method devised by Dr. Edmund Prince Fowler Sr. for the determination of percentage loss of usable hearing is presented and described. Grove stated that both these methods are a result of the intensive work done by the Committee of Consultants on Audiometers and Hearing Aids of the Council on Physical Therapy of the American Medical Association. They have taken into account several factors in the establishment of a base line for total loss of hearing for speech; they consider the most important part of the hearing range for industrial purposes; hearing with a loss of less than 10 decibels is classified as normal; weighting of percentage losses is given more value in the primary speech area, of 512 and 2048 cycles, than in areas above and below it; finally, they recognize that a hearing defect becomes progressively greater as one goes from the threshold toward the base line for total loss of intelligible

15. Sabine, P. E.: On Estimating the Percentage Loss of Useful Hearing, Tr. Am. Acad. Ophth. (1941) **46**:179 (March-April) 1942.

speech. Although the method of approach is decidedly different with the two standards, Grove believes that computations by either method will be essentially the same. One of these two methods will probably be presented to the medical profession soon by the Council on Physical Therapy.

The Consultants on Audiometers and Hearing Aids of the Council on Physical Therapy[16] present the following tentative standard procedure for evaluating the percentage of useful hearing in medicolegal cases. This method has been adopted by the Council on Physical Therapy and by the Section on Laryngology, Otology and Rhinology of the American Medical Association. The procedure is based on measurements of hearing loss with an accepted standard audiometer. It is also based only on threshold measurements of acuity of hearing for air-conducted sound. The following decibel losses, as measured by an accepted standard audiometer, represent a 100 per cent loss of useful hearing: 128 cycles, 65 decibels; 256 cycles, 80 decibels; 512 cycles, 85 decibels; 1024 cycles, 95 decibels; 2048 cycles, 95 decibels, and 4096 cycles, 90 decibels. A chart presents the percentage losses corresponding to measured decibel losses in the octave intervals. To evaluate the percentage loss of hearing the chart is used in the following manner: Hearing losses for the different frequencies in each ear are plotted in the usual manner. Successive points are connected by straight ruled lines. The percentage loss to be assigned to each octave interval is the figure in the space immediately above the straight line drawn in the octave. Where two or more spaces are intersected, the average of the figures in the intersected spaces is taken. The figures for the four octaves for each ear (256, 512, 1024 and 2048) are set down in their respective columns, headed "Right Ear" and "Left Ear," at the right of the chart. The sum of the figures for each ear is the total percentage loss of hearing for that ear. To compute the combined percentage loss of hearing for the two ears, instructions are given at the bottom of the chart. To seven times the total percentage loss for the better ear is added the total percentage loss for the worse ear and then the sum is divided by 8. This represents the average of the losses for the two ears taken separately in the ratio of 7:1. The weighting of percentage loss shown by the better ear as compared with that shown by the worse ear of 7:1 represents the composite judgment of the committee based on both clinical experience and practical considerations.

The procedure is recommended as a tentative standard based on present knowledge of the relation between acuity of hearing measured

16. Tentative Standard Procedure for Evaluating Percentage of Useful Hearing Loss in Medicolegal Cases, Special Reports, Arch. Otolaryng. **36**:590 (Oct.) 1942.

by the audiometer and the ability to interpret direct speech in a familiar language. This procedure is subject to revision when further authoritative data on the problem become available.

The charts, printed on 4 by 5 inch cards, may be purchased by writing the Secretary of the Council on Physical Therapy.

HEARING AIDS

A complete list of hearing aids and audiometers accepted by the Council on Physical Therapy of the American Medical Association as of July 1942 is given by Macfarlan.[17] The trade name, the model, the manufacturer's name and address and the reference to the report in *The Journal of the American Medical Association* of each of the instruments are included. This is the first comprehensive list of hearing aids to be published. For more details one should refer to the complete report of the aid. The following hearing aids are acceptable to the Council: Acousticon, several models; Aladdin; Aurex, semiportable; Maicophone; Mears aurophone; Otarion; Radioear, several types; Ravox, semiportable; Telex, several models; Vacolite, two models, and Western Electric. The audiometers accepted are the Maico model D-5 and the Western Electric, models 6B and 4C.

Berry [18] presents the case for the hearing aid by comparing the results of several of the leading advocates of the fenestration operation with benefits derived from hearing aids approved by the Council on Physical Therapy and reported on by leading acoustic engineers. From this investigation, he concludes that the well fitted modern hearing aid can give greater amplification than the fenestration operation to a wider group of hard of hearing persons at less cost in expense, pain and worry. The average gain from the fenestration operation in selected cases reported by men like Shambaugh, Lempert and Campbell ranges from 21.8 to 25.7 decibels, as compared with an average gain of 43 decibels in a selected series of hearing aid fittings. Berry mentions several valid reasons for the fenestration operation. It is a skilled procedure, which offers a challenge to the otologist and makes him prepare himself adequately. It offers a means by which a patient may avoid advertising his infirmity by wearing an aid. Most important, it is claimed that the operation stops the advance of progressive deafness. This claim should have further study, for if it is valid, it is an important argument and accomplishment for the fenestration operation. Berry recommends that when a fenestration operation is advised, the patient in all fairness be

17. Macfarlan, D.: Hearing Aids Accepted by the Council on Physical Therapy, Arch. Otolaryng. **36**:240 (Aug.) 1942.

18. Berry, G.: The Case for the Hearing Aid, Laryngoscope **52**:615 (Aug.) 1942.

informed more accurately on the performance of a properly fitted modern hearing aid. He also suggests that the acoustic engineer devise a more accurate method of fitting an aid. The otologist should examine carefully every patient who contemplates the wearing of a hearing aid, give necessary treatment when indicated, make the plaster ear mold and check the selected instrument's accomplishments for the patient. He should also rely on the acoustic expert to make the careful selection and the detailed adjustments of the instrument.

Bunch [19] feels that a certain technical knowledge of a hearing aid is prerequisite for the proper fitting of the aid. A graph giving the amount of amplification in decibels at semioctave intervals of C from 256 to 4096 double vibrations and information regarding peaks or valleys between these points, if present, are required from the manufacturer by the Council on Physical Therapy. This information is available through the Council or may be obtained direct from the manufacturer. Knowledge of the patient's residual hearing as shown by the audiometric tests and an accurate idea of the hearing aid's performance will greatly simplify the proper fitting of hearing aids.

Bunch discusses the amplification characteristics of nine hearing aids which were tested by an engineer at the request of the Council on Physical Therapy. He shows the difficulties that would arise in applying these aids to different types of audiograms. Their differences of performance show how impractical it is to apply any one type of hearing aid to all types of impairment of hearing.

Bunch also claims that a manufacturer given an audiogram cannot, as a rule, select a hearing aid to compensate accurately the losses shown in the audiogram. The research of the manufacturers is primarily concerned with problems solution of which will lead to the production of an instrument which will benefit the greatest number of persons. Such an instrument is manufactured with an added tone control which will alter the amplification characteristics at certain frequencies. If a person should have a greater loss for high tones than for low tones, the tone control will be set to amplify high tones more than low ones. In this way it is possible to fit more or less accurately one stock instrument to a fairly large number of prospective wearers. As long as one instrument is made to fit a large number of persons with different hearing losses, it is certain that some will be fitted better than others, and this will eventually react against the industry as a whole. Unless a large stock of instruments with a wide range of amplification characteristics is on hand, or can be sent for, this difficulty cannot be overcome.

The price of the instrument is also discussed. Bunch believes that the cheapest reliable hearing aid should be recommended to the patient.

19. Bunch, C. C.: Hearing Aids, Tr. Am. Acad. Ophth. (1941) **46**:163 (March-April) 1942.

Operating costs should also be considered. A table showing the variations in voltage and current consumption of nine vacuum tube hearing aids accompanies the article. Bunch also believes that the preparation of the ear mold should be under the guidance of an otologist.

A properly fitted hearing aid will enable a person to hear and understand conversation. This is the real test for the aid. It is not necessary for a person to have to wear an aid for a time before he is able to understand conversation. The usual articulation tests with use of spoken words, sentences or nonsense syllables to determine the efficiency of a hearing aid are of value only if all variables in the test are eliminated. Some of these variables are the pitch of the voice, the distance from the microphone and the type of hearing impairment.

Bone conduction receivers can be selected only on the basis of chance until some standardized method is devised for measuring in absolute units the acuity of hearing by bone conduction. Only those persons having good hearing by bone conduction can possibly benefit by a bone conduction hearing aid. From an electromechanical standpoint a hearing aid of the bone conduction type is less efficient than one of the air conduction type. A bone conduction aid must supply power to vibrate the entire head, whereas an air conduction aid needs only sufficient power to vibrate the column of air between the diaphragm of the receiver and the vibrating mechanism of the ear. Bone conduction receivers seemed to be the only solution for the hearing aid problem when air conduction aids gave inadequate and insufficient response to voice sounds. This is no longer true with the modern vacuum tube aids.

Langenbeck [20] presents the possibilities and limitations of a modern hearing aid for which he claims amplification true up to 10,000 cycles without adventitious noises. The instrument has been brought down in size to 20 by 28 by 6 cm., which is that of a brief case, and he hopes after the war to have it produced in a more convenient size, as the technical difficulties are not unsurmountable. More than the necessary intensity can be obtained without any distortion or adventitious noise, which is not true of the electromagnetic ear pieces or bone conduction receivers now in common use. Amplification is especially necessary for frequencies from 4000 to 8000 cycles in cases of inner ear deafness to permit the hearing of the formants of speech making up certain of the consonants. This has proved useful in the education of children in schools for the deaf. An audiogram ranging below 80 decibels is shown. The patient could understand only loud voice at the ear, which is the poorest hearing for which this instrument is useful. Below this level any kind of instrument will do, but of course is not satisfactory. The electromagnetic type will give greater amplification, but not the dis-

20. Langenbeck, B.: Possibilities and Limitations of Hearing Aids, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **149**:199, 1941.

crimination or the freedom from noise. The author cites the case of a teacher with otosclerosis who conducted his classes successfully by placing an instrument on his desk, and the case of a patient with a loss of 70 decibels who with the instrument could hear whispered voice at 6 meters.

Patients with an average loss of 70 decibles who hear loud voice only at the ear, but who may hear at 6 meters in an acoustically insulated room, do not hear in a lecture hall and are accustomed to hearing only fragments of conversation. Such persons may require reeducation in understanding consecutive speech and practice in hearing in halls of various sizes. They must also get used to the ordinary surrounding noises, which they have long forgotten and which the normally hearing person ordinarily disregards. In other words, not only is it necessary to give the patient elective amplification so that he hears most of the formants of speech, but he must learn how to use his reestablished possibilities of hearing.

For the less severely deaf patient, who has not forgotten how to hear, this instrument brings an effortless understanding of speech and is essential for economic reasons.

No technical description of the instrument accompanies this article.

CONSERVATION OF HEARING

Bunch [21] brings up two questions in the discussion of conservation of hearing. He asks, first, whether it is generally recognized that hearing losses result from certain industrial hazards and, second, whether anything can be done and is being done to prevent them. He states that many employers fail to take advantage of having the hearing of their employees tested, possibly because of the fear of additional legal regulations and of possible questions of compensation arising therefrom. Employees also are fearful of having this done because they think it will bring about loss of employment. Factors which produce loss of hearing in industry are exposure to unfavorable weather conditions, producing infections of the ear and secondary hearing losses, sudden wide variations in atmospheric pressure, such as are experienced by caisson workers and aviators, and exposure to loud noise. The hearing loss in persons exposed to noise primarily affects the upper tone range, as reported by other observers, and later involves the lower frequencies. Many persons show no impairment in hearing for the spoken voice because the speech range is not involved. The vowel sounds have complex patterns of tones between 300 and 3000 double vibrations. The frequency of most of the consonant sounds is somewhat higher. A

21. Bunch, C. C.: Conservation of Hearing, J. A. M. A. **118**:588 (Feb. 21) 1942.

person having a pronounced hearing loss for tones above 2048 double vibrations may have no apparent difficulty in understanding conversation. Bunch reports cases of this type of defect in a foundry "chipper," a boiler maker–riveter, a telephone operator, policemen who do target practice, a farm tractor operator and a whippet tank driver. He discusses the effectiveness of cotton plugs in the ear. The cotton plugs cause little lowering of the threshold for low tones, but block out high tones more effectively. Petrolatum-cotton packs obstruct both low and high tones consistently to a greater extent than do the dry packs. If, as reported by Perlman, the sounds of low frequency produce less acoustic trauma than those of high frequency, then petrolatum-cotton packs may be effective in preventing deafness. Bunch makes a plea for determining the status of hearing in employees at the beginning of employment. This would protect both employee and employer, and conditions which are harmful would be determined by repeated tests and suitable protective devices could be installed. It would provide adequate insurance for the employer against employees who may claim damages for losses in hearing which existed prior to the time of employment. Much scientific information concerning otologic problems could also be gathered from these tests. The question of compensation is now being considered by the Council on Physical Therapy of the American Medical Association. It is hoped that some uniform scale for compensation for hearing loss will be developed and universally adopted.

Chamberlain [22] presents a preliminary report of the results of audiometric examinations of 4 boiler makers before and after exposure to noise. He finds that aural fatigue in boiler makers may be great and the power of recovery remarkable. However, examinations at variable periods after exposure to noise show that recovery does not always take place within fifteen hours of rest from the noise. The ears of one person may be more susceptible than those of another to the same fatiguing noise. The high tones are usually affected much more than the low ones. However, if there is a permanent loss for high tones before exposure to noise, the lower tones may be the only ones for which there is significant fatigue. Adequate intervals of rest between periods of exposure to noise might prevent or delay the onset of permanent cochlear damage.

Almour [23] presents the clinical picture of the "blocked ear" found in caisson workers, which he considers an entity. There are several variations, dependent on the degree of involvement. In 46 cases there was some immediate impairment in hearing. This loss showed no

22. Chamberlain, D.: Occupational Deafness: Audiometric Observations on Aural Fatigue and Recovery, Arch. Otolaryng. **35**:595 (April) 1942.

23. Almour, R.: The "Blocked Ear" of the Caisson Worker, Laryngoscope **52**: 75 (Jan.) 1942.

characteristic for this type of injury. In 19 cases the loss was in the 2048, 4096 and 8192 octaves, and in 21 cases, in the 128, 256 and 512 ranges. In 1 of the remaining 3 cases, there was a parallel loss for air conduction in the three upper notes, and in the other 2 cases, a loss in one ear for the lower octaves and in the other ear for the higher ones. In the vast majority of cases the hearing returns to within normal limits after measures are taken to reestablish the patency of the eustachian tube. Diathermy aids in absorbing the hemorrhages. Almour concludes that the effect of compressed air on hearing needs intense study. As yet the estimation of compensability on the basis of percentage of hearing loss due to a blocked ear is not possible. He suggests that legislative measures should be enforced to compel workers with such disability to have an accurate estimation of their hearing made before employment so that otologists will have further material in evaluating this condition.

Grove [24] reports on 43 replies received to a questionnaire sent to employers of labor of all types to determine whether impaired hearing is a great handicap to any particular types of work. He concludes that with the exception of a few special occupations, such as switchboard work, stenography, proofreading and salesmanship, there is no type of work that the hard of hearing person cannot do well. However, to accomplish this he must approximate his psychologic attitude to the normal, compensate for his defect with lip reading and hearing aids, make himself just a little more efficient than his fellow and be sober, industrious and honest. Industry will employ him in spite of his hearing defect.

In another article [25] Grove presents the report of the United States Department of the Interior, bulletin 13, 1936, on employers' rating as to occupational success of the deaf and the hard of hearing. The ratings were made under four headings: persons succeeding very well, 54.1 per cent; persons succeeding fairly well, 32.0 per cent; "getting by," 11.2 per cent and persons, failing 2.7 per cent. Employees with a better education or with training for a special trade got along better than those without.

Wells [26] discusses the various causes of deafness. The hereditary factor plays an important part in otosclerosis and congenital deaf-mutism. He cites the work of Fay, who analyzed 4,000 family histories and pointed out that although no deafness may be evident in the immediate parents of a deaf child, an undiscoverable defect is present and will crop out in the regular mendelian proportion. When both parents are

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24. Grove, W. E.: Does Your Job Depend on Hearing? Hygeia **19**:895 (Nov.) 1941.

25. Grove, W. E.: Proceedings of Central Zone Conference, American Society of Hard of Hearing, June 1941, p. 69.

26. Wells, W. A.: Deafness: A Consideration of Its Causes, Am. Ann. Deaf **87**:212 (May) 1942.

congenital deaf-mutes, there will be a deaf-mute offspring in every sixth or seventh case. When only 1 parent is a deaf-mute, the incidence of a deaf-mute offspring is about 1 in every 30 cases. Hearing tests made on school children have shown that environment and climate are also factors. The incidence of deafness is greater in children of poorer sections than in those of well to do areas. Climates having cold, damp weather produce more aural trouble, which may lead to impairment of hearing. Age is also a factor. Infections of the middle ear are more common in young children. Otosclerosis has a predilection for the important years of early manhood and womanhood. Finally, hearing normally drops with advancing age, from 20,000 cycles as the upper limit at 20 years of age to 8,000 cycles at 80 years of age. The general state of health may have some bearing on the hearing. Systemic diseases, such as diabetes, arteriosclerosis, tuberculosis and syphilis, may affect the auditory apparatus. Focal infections may produce changes in the auditory nerve. Occupations of certain types have a deleterious effect on the organ of hearing. Outdoor work and exposure to severe cold and bad weather are factors. Severe variations in air pressure are injurious, as in the case of caisson and tunnel workers. Noise is an important factor and may be classified according to the following causes: animate nature; war; building and construction; traffic and transportation; manufacture; commerce and communication. Chemical poisons also play an important part in deafness. Drugs may be transmitted to the unborn child by the mother and cause congenital defects in hearing. Important substances are quinine, salicylates, mercury, arsenic, lead and carbon disulfide. Other agents harmful to the auditory nerve are coffee, alcohol and tobacco. Infections and contagious diseases may produce secondary aural complications and eventual impairment of hearing. The common cold, adenoids, diseased tonsils and sinal infections, likewise, may be responsible for hearing defects.

To understand more clearly the mechanism of inheritance of deafness, Hughson and associates [27] made a study of the families of 307 children of the Pennsylvania School for the Deaf. The children were first classified as to whether they were actually born deaf, embryonic deafness, or whether the trouble came on later, nonembryonic deafness. In addition to the history, the criterion was whether the child ever used articulated speech without any special instruction. With this criterion, it was found that 35.8 per cent of the school population had embryonic deafness, or deafness which occurred before the first year of life and was not due to injury or disease at birth or afterward. The embryonic type of deafness further manifests obviously a familial tendency, whereas the

27. Hughson, W.; Ciocco, A., and Palmer, C. E.: Studies on Pupils of the Pennsylvania School for the Deaf, Arch. Otolaryng. **35**:871 (June) 1942.

nonembryonic type does not exhibit this tendency. The authors' observations also show that the embryonic type of deafness is transmitted from parent to offspring by a single recessive gene. This means that if both the man and the woman have embryonic deafness their offspring will be deaf. If only one parent has this type of deafness, either one half or none of the children will have embryonic deafness, depending on whether the parent with good hearing is a carrier of or is completely free from the gene in question. If one of the parents, although having good hearing, is a carrier of the gene, either 25 per cent or none of the offspring will have embryonic deafness, depending on whether the other partner with good hearing is also a carrier. From the analysis, it seems that the deafness is transmitted according to the mendelian pattern, involving a single recessive gene. If this hereditary influence exists, then, whether or not a history of deafness in the immediate family is elicited, a person with embryonic deafness will transmit the particular gene to his offspring, and the chances that the transmission will be manifest in the offspring will depend on the hereditary makeup of the spouse. Even though a person may have good hearing or an acquired type of deafness, if embryonic deafness is present among the brothers or sisters, one must be suspicious that that person is a carrier of the recessive gene for deafness.

Buckman [28] reports on the Pennsylvania plan for hard of hearing school children which is now being used in Bethlehem, Pa. This plan was reported by us in a previous review. Under the plan, there is a direct tie-up between the public health authorities and the active medical profession in the study and care of this problem. A letter is sent out to every physician in Bethlehem explaining the need for early diagnosis and proper treatment of impaired hearing in children and assuring members of the medical profession that every effort to cooperate will be made on the part of the public health and school authorities, as well as the parent-teachers' associations, to bring this about. Likewise, letters are sent to the parents telling them what is being done and urging proper attention when hearing defects are found. The school nurse follows up these letters when treatment and examination have been suggested. A phonograph with an electric pick-up employing a record of constant level set at 10 decibels above threshold is being used. Each child is examined separately with a pair of ear phones employing a switch for selecting the right or the left ear. The child has only to answer the numbers he hears. One child can be examined in from thirty to forty-five seconds. The plan has been working out satisfactorily in Bethlehem. No complaints have been received as to lack of medical

28. Buckman, L. T.: Early Recognition of Deafness: The Pennsylvania Plan for the Hard of Hearing School Child, Pennsylvania M. J. **45**:345 (Jan.) 1942.

care or its efficiency. Each examiner has an audiometer, and when a child with impaired hearing is discovered the examiner makes a more particular study to find a possible cause.

Nash [29] discusses the importance of residual hearing in children, especially from the standpoint of education and speech development. He defines residual hearing as that amount of hearing that remains after there is a degree of lasting impairment. Its common interpretation is the hearing of persons with severe impairment and little or no understanding of speech. A general classification of hearing loss and the training aid required, according to O'Connor, is as follows: Children with a 0 to 20 decibel loss require only a favorable seat in a regular school; those with a 20 to 30 decibel loss may require in addition some lip reading; a 30 to 40 decibel loss requires a favorable seat, hearing aid, speech correction and special tutoring; a 40 to 50 decibel loss requires, in addition partial attendance in a special class in a regular school; a 50 to 60 decibel loss requires full attendance in a special class in a regular school, full attendance at a special day school for the acoustically handicapped or full attendance at a residential school for the deaf; a 60 to 100 decibel loss requires full attendance in special classes in a regular school or full attendance at a residential school for the deaf. Kerridge's classification is as follows: children hearing conversational voice at 20 feet (6 meters) require only a favorable seat in a regular school; those hearing conversational voice at more than 2 feet (60 cm.) and at less than 20 feet require attendance in a special class for the acoustically handicapped in a regular school, and those hearing conversational voice at 2 feet or less should have full attendance in a school for the deaf.

In comparing hearing loss with speech development, Kerridge has the following classification: Persons with a loss of 0 to 30 decibels have natural speech; those with a loss of 30 to 50 decibels have natural or partially defective speech; those with a loss of 50 to 90 decibels have fair or poor speech, and those with a loss of 90 to 100 decibels have poor speech.

The records of 218 pupils enrolled in the Rochester School for the Deaf are analyzed. In the group having a hearing loss of 30 to 45 per cent much depends on the character of the graph. If the graph descends at high frequencies there is difficulty in interpreting spoken language, and speech will be defective if the impairment is congenital. The group having a loss of 45 to 65 per cent are wholly unable to interpret spoken language without training unless they acquired language before the onset of deafness. In the training of the group having a hearing loss of 65 to 100 per cent the principal purpose is to stimulate the sense of hearing sufficiently for the child to acquire some vowel discrimination

29. Nash, C. S.: Residual Hearing, Laryngoscope **52**:577 (July) 1942.

and to recognize to a certain extent the pitch, rhythm, inflection, phrasing and emphasis used in spoken language. Twelve teachers from the faculties of seven of the country's foremost schools for the deaf have come to the following conclusions regarding the value of residual hearing: Children may benefit from the use of residual hearing as a medium for interpreting spoken language, for acquiring new language through speech, for speech correction and for development of more intelligible and natural speech. These teachers also stated that benefits from residual hearing depend also on the type of deafness, the frequencies heard, the intelligence, the age at onset of deafness, the emotional stability and the health of the child. They conclude after these factors are taken into consideration that a child with as little as 20 or 25 per cent of residual hearing may make notable improvement in his speech pattern. They recognize also that the smallest amount of residual hearing may be of some possible use.

An investigation of the acoustically handicapped children of the public schools of the city of New York was made by a committee. Hunt [30] presents some of the facts obtained through a study of 1,080 sample cases. The breakdown analysis showed 401 cases of suppurative healed otitis, 394 cases of nonsuppurative healed otitis and 125 cases of suppurative chronic otitis, with one or both ears discharging at the time of examination. There were only 54 cases of nerve deafness. The etiologic factors contributing to the otologic condition were as follows: hypertrophied or diseased tonsils or adenoids, 405 cases; sinal infection, 199 cases; lymphoid tissue, 190 cases; pathologic condition of the turbinates, 189 cases, and deviated obstructing septums, 15 cases.

The study indicates a definite problem in the adaptation of acoustically handicapped pupils to their physical teaching surroundings, to the teaching approach to and the physical aid for their handicap. It shows also the importance of otologic examination in the solution of this problem. It indicates, moreover that there is a real problem in preventive deafness in the public schools.

Goodfellow [31] reports on the reeducation of persons with defective hearing. Since many investigators have observed that some hard of hearing persons make much more effective use of their residue of hearing than others and since it is clinical experience that this cannot be accounted for on the basis of intelligence, the effects of reeducation by various procedures have been studied. The Northwestern University speech perception test, which consists of 100 nonsense syllables employing the speech sounds most frequently used in spoken English and prescribed

30. Hunt, W. M.: What Is Being Done for the Deafness of School Children? Laryngoscope **52**:230 (March) 1942.

31. Goodfellow, L. D.: The Re-Education of Defective Hearing, J. Psychol. **14**:53 (July) 1942.

from a phonograph recording, showed no significant correlation between a person's loss of hearing and his score on this test. This test, however, was limited to persons having a hearing loss between 30 and 65 decibels, which might explain the lack of correlation found in the data. A group of 6 subjects were taken for study. The training program consisted of four factors: adaptation to frequency distortion; clarification of phonetic concept; direction of attention to secondary cues contained in the gross pattern of speech, and development by the deafened person of an objective attitude toward his handicap. This method was highly successful with the 6 persons studied. Further research, however, is necessary to determine the precise role of the various factors and the best technic for employing them. This investigation brings out several points in regard to hearing aids. If reeducation enables a person to utilize better his residual hearing, it may be necessary to correct his hearing loss gradually in order to reeducate him to hear normal speech. Also, the time when a hearing aid should be prescribed cannot be entirely determined by the audiogram alone. A test for speech perception may furnish the necessary data. The real question may not be how deaf a person is but, rather, how well he uses his marginal residue of hearing.

Burchard and Myklebust [32] have made a study of the effects of congenital and adventitious deafness on intelligence, personality and social maturity. Two groups of children having congenital and adventitious deafness, respectively, were found to be of average intelligence. The deaf boys seemed to rate slightly superior to the deaf girls. A comparison of the children who had been in residence in an institution for more than four years with those who had been in residence less than that time revealed no significant difference. The investigation revealed retardation in social maturity in both types of deaf children, and those in residence in an institution were as immature as the others. The authors conclude that residential schools are not adequately training children in social competence. Their study of personality revealed that both types of deaf children exhibit more behavior problems and problem tendencies than the normal-hearing children. The type of deafness is not as important as the deafness itself from the point of view of educational rehabilitation. Boys exhibit much more of the problem type of behavior than girls. Residence in a school did not appear to alter the condition. This observation points to the need for much more psychologic work in residential schools.

Criswell,[33] in discussing impairment of hearing in the aging, emphasizes the importance of prophylaxis. He mentions the various forms of

32. Burchard, E. M. L., and Myklebust, H. R.: Comparison of Congenital and Adventitious Deafness with Respect to Its Effect on Intelligence, Personality and Social Maturity, Am. Ann. Deaf **87**:140 (March); 241 (May); 342 (Sept.) 1942.

33. Criswell, J. R.: Symposium on the Medical Problems of the Aging, Hahneman. Monthly **77**:236 (April) 1942.

such loss: senile, progressive catarrhal deafness; otosclerosis; impairment following suppurative otitis media, and deafness due to acute infectious diseases, some of the general diseases, such as arteriosclerosis, diabetes, nephritis and syphilis, focal infection, drugs and noise. He suggests that in all cases infectious disease and suppurative otitis media be properly diagnosed and treated, focal infections be sought for and removed, careful hearing tests be made when the deafness is progressive and lip reading or the proper selection of a hearing aid when necessary be employed early.

Loch [34] investigated the effect of simple occlusion on hearing. He performed the experiment on himself by inflating a soft rubber balloon in the nasopharynx to obstruct the tubes. The hearing was tested before and after occlusion at various intervals over a period of one and a half hours. This occlusion caused, first, a loss in the region of high tones. Later the hearing for middle and low tones became slightly impaired, but never to as great a degree as that for high tones. The hearing returned to normal, or where it was before the occlusion, immediately after restoration of adequate ventilation of the middle ear. This was accomplished by removal of the balloon, the Valsalva procedure and swallowing. The prompt recovery of hearing acuity shows that the impairment was a result of pressure changes in the middle ear rather than of secondary changes, such as edema of the organ of Corti or of the round window membrane. These experiments confirm the views of Crowe and Guild, who showed that an uncomplicated tubal occlusion causes a hearing loss for high tones, sometimes followed by impairment for other tones also. This again emphasizes the importance of early recognition and treatment of patients with this type of impairment of hearing.

Wever [35] studied the problem of the tonal dip by examining a large series of serial sections of the temporal bones of persons whose audiograms indicated a pronounced local depression of sensitivity. The study revealed that there was a large preponderance of dips among males and that the dips in nearly all cases were at a frequency of 4096 cycles. No region was identified as specifically responsible for the local depressions. There was a somewhat higher degree of pathologic change in structures of the inner ear in cases in which large dips were present. The various theories as to the cause of the tonal dip are discussed. It appears that the 4096 cycle dip is due to impaired function of the organ of Corti in a region of the lower basal half-turn, although no such impairment is

34. Loch, W. E.: The Effect on Hearing of Experimental Occlusion of the Eustachian Tube in Man, Ann. Otol., Rhin. & Laryng. **51**:396 (June) 1942.

35. Wever, E. G.: The Problem of the Tonal Dip, Laryngoscope **52**:169 (March) 1942.

revealed by the microscopic technic employed by the author. Possibly improved methods of histologic treatment may provide a final answer to the problem.

Wever and associates [36] determined the effects of pressure on the ear of a cat. Their experiments show that an increase in the air pressure of the cavity of the middle ear has a pronounced effect on the electrical responses of the cochlea. In general, low tones are affected more than high tones, but the relation is not exactly in the order of frequency. Effects for bone conduction were also obtained, but differed from those for air conduction. When the ossicular chain is broken and bone conduction is used, the effects of pressure are comparatively small. The authors conclude that pressure in the middle ear has its principal effect on the ear drum, though minor effects arise also in its action on parts of the inner ear.

36. Wever, E. G.; Bray, C. W., and Lawrence, M.: The Effects of Pressure in the Middle Ear, J. Exper. Psychol. **30**:40 (Jan.) 1942.

(To Be Concluded)

Abstracts from Current Literature

Ear

THE PSYCHOLOGY OF DEAFNESS IN CHILDREN. GEORGE L. DRENNAN, Illinois M. J. **79**:227 (March) 1941.

Defective hearing imposes mental strain, isolation and loneliness on the deaf and hard-of-hearing child and arrests his intellectual development. His continual struggle to make himself understood may result in a marked inferiority complex. Many of the psychologic reactions to deafness may be helped. The prevention of those diseases which produce deafness is most important.

BARBOUR, Peoria, Ill. [AM. J. DIS. CHILD.].

ELECTROKINETIC CHANGES IN THE ENDOLYMPH AS A HYPOTHETICAL CAUSE OF FALLING AND PAST-POINTING DUE TO STIMULATION BY GALVANIC CURRENT. LEWIS J. POLLOCK, ISIDORE FINKELMAN and I. C. SHERMAN, J. Nerv. & Ment. Dis. **93**:473 (April) 1941.

Although it has been known for a long time that the passage of an electric current through the head produces vertigo and a tendency of the subject to fall and past point toward the anode, controversy still exists as to the point and manner of action of the current. Pollock and his associates point out that it is unlikely that the current stimulates the vestibular nerve directly because these reactions do not occur on application of an alternating current such as is effective in stimulating other nerves in the body and because the calculated chronaxia of the falling reaction is much greater than that of other nerves. In addition, the falling reaction occurs only on the continuous passage of current and not on make or break. In a new series of experiments Pollock and his associates confirmed these facts and discovered further that a unidirectional pulsating current with a frequency of 15,000 per second caused past pointing toward the anode. Such a current produces stimuli with a duration of only three hundred-thousandths second, suggesting a degree of irritability unlikely in the vestibular nerve. They concluded, therefore, that the passage of a current through the head does not stimulate the vestibular nerve directly but produces some physical change in the endolymph. That this change is not the localized production of heat or electrolysis is suggested by the fact that the average amperage of a pulsating current required to produce falling and past pointing varied with the duration of the individual pulsations. On the other hand, the relations of current, duration of stimuli and interval between stimuli found in the production of falling or past pointing are the same as those described for an electrophoretic displacement of solid, liquid or gaseous particles in a fluid medium. This fact suggests to the authors that an electric current passing through the head produces vertigo, falling and past pointing by inducing such an electrokinetic change in the endolymph and not by stimulating the vestibular nerve.

MACKAY, Chicago [ARCH. NEUROL. & PSYCHIAT.].

PSYCHOLOGIC ASPECTS OF DEAFNESS: A DISCUSSION H. FREY AND OTHERS, Proc. Roy. Soc. Med. (Sect. Otol.) **34**:309 (April) 1941.

A study of the psychologic conditions in people with defective hearing, Frey stated, would help to a more complete understanding of their complaints and would be of assistance in alleviating these. From the literature he learned that among deaf-mutes the uneducated often appear queer and not easy to handle, while the educated ones are often careful and vigilant, concentrate well and do good work

in many trades. He thought that most deaf-mutes were not quite normal apart from their hearing defects, that the congenitally deaf ones were degenerate, showing abnormalities of their central nervous system, of their intelligence and of their psychic disposition as well as some somatic stigmas. If, however, deafness was acquired in early life, it could be assumed that the central nervous system had not survived the strain of a disease such as meningitis without lasting inferiority of function. He was of the opinion that the deafened person as a rule overestimates the extent of the deafness and many overestimate the practical consequences of it. The patient with otosclerosis represented a special type of a highly nervous character with highly unstable vasomotor conditions. He thought the mental depression of the deafened person was open to relief by the application of mental energy and effort and that this opened a wide vista for psychic treatment and reeducation.

Dr. A. B. Stokes discussed the psychologic association of organic disabilities in general and its application in the instance of deafness.

Mrs. T. R. Ewing analyzed various phases of the psychologic aspects of deafness by specific illustrations. She stated that the psychologic effects of deafness, in addition to deafness itself, depended on such factors as the health and the age of the patient, his environment and experience, his social attitude, his disposition toward difficulties, his intelligence, the nature of his work, his resourcefulness and his sense of humor.

CAMPBELL, Philadelphia.

RECURRENT MASTOIDITIS. FRANCISCO H. RIVERO, Arch. venezol. Soc. de oto-rino-laring., oftal., neurol. **2**:37 (March) 1941.

Rivero discusses recurrent mastoiditis and differentiates it from residual mastoiditis.

In cases of the former the original mastoidectomy produced a complete cure. This implies that the middle ear was dry, the tympanic membrane was completely healed and had resumed its normal color and position, the postauricular wound was healed, and the scar was resuming its normal color and texture. The underlying altered tissues underwent regeneration, the first step of which is fibrosis. If sufficient time had elapsed, through osteogenesis, there was replacement of true bone. This, Rivero believes, can again assume the normal architecture of the mastoid process, reforming a true antrum, periantral cells and frank cellular structure throughout the entire mastoid process. The longer the interval before reinfection, the greater the amount of bone formation and the greater the extent of restoration of this normal cell structure. There is divergence of opinion as to whether this rebuilding of cell structure originates from within the mastoid operated on and some of the residual intracellular septums or from the periosteum.

In cases of residual mastoiditis, on the other hand, there never has been complete cessation of discharge, either from the middle ear or from the postauricular wound, because of incomplete mastoidectomy, improper postoperative care, negligence or improper attention to the original infecting foci (nose, throat, sinuses, tonsils) or the general lowered resistance of the patient.

Mastoiditis may recur at any time from several weeks to many years after complete healing of the original infection. The factors favoring recurrence are often the same factors that caused the original infection. The author emphasizes the fact that the initial infection of the mastoid does not immunize the patient against recurrences. Some of these factors can be found in the nose, throat and sinuses, where residual infections have not been completely eradicated and some in systemic or hereditary disease of the patient or acute infectious diseases.

In closing, Rivero stresses the psychologic attitude of the patient toward the surgeon. Frequently the patient assumes the attitude that it had been due to either carelessness or an incomplete operation that the mastoid disease recurred. This lack of confidence in the surgeon is particularly evident when the recurrence appears within a relatively short time after the original mastoidectomy.

PERSKY, Philadelphia.

CONGENITAL AUDITORY ATRESIA: ITS PLASTIC REPAIR WITH FUNCTIONAL RESTORATION OF HEARING AND PHONATION IN THE DEAF-MUTE. FELIX VEINTEMILLAS, Artistica, April, 1940.

Veintemillas describes a method of plastic repair for congenital atresia of the ear in which he made a postauricular incision through the skin and retracted the tissue forward to expose the temporal bone in the region of the mastoid. Using either a trephine or a chisel, he made a funnel-shaped or cylindric canal into the more or less rudimentary middle ear. The canal had the promontory as its base. Then, utilizing the skin around this opening, he made a cutaneous flap, extending into the new canal, which insured both the preservation and the epidermal lining of the canal. The residual auricular or cartilaginous appendage was moved backward, sliding over the new-formed canal like a bridge, and was sutured in this position so that the canal was definitely anterior to the appendage and resembled a normal canal.

He found that deaf-mutes with this condition were successfully cured provided that the operation was performed at a suitable age. His best results were obtained in those between the ages of 5 and 12 years. Prior to the fifth year he encountered difficulty in obtaining satisfactory results. This was due to the incomplete development of the temporal bone and mastoid process, which afforded only a limited operative field. After the twelfth year the results again were not very favorable, owing to functional progressive atrophy of the acoustic nerves. His patients required reeducation so that they could learn speech in association with the recovery of the hearing. This necessitated prolonged conscientious education in speech. He reports 8 patients in whom 12 reconstructions were made. His first patient, operated on in 1922, enjoyed a complete recovery of hearing and speech.

PERSKY, Philadelphia.

Pharynx

CHEMOTHERAPY IN THE MANAGEMENT OF LUDWIG'S ANGINA. JOHN F. CONWAY, J. A. M. A. **118**:953 (March 21) 1942.

The author discusses in moderate detail the important points of Ludwig's angina, the etiologic factors, the symptoms and the treatment.

Three cases are reported in which the condition fell within the specifications of Ludwig's angina and in which the author employed sulfanilamide or one of its derivatives, especially sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole). Recovery was brought about without surgical intervention.

In the first case the following treatment was employed: Continuous moist dressings were applied to the neck, and continuous steam inhalations were given. A tracheotomy set was held in readiness. Fluids were supplied intravenously. Five intramuscular injections of 5 cc. of a 5 per cent solution of azosulfamide were given in the first twenty-four hours. Sulfathiazole, 120 grains (8 Gm.), was given rectally soon after admission, and within the next thirty-six hours an additional 120 grains of the drug was given by the same route in 60 grain (3.9 Gm.) doses. The following day the patient was able to swallow and began to take fluid by mouth; she was given 15 grains (0.98 Gm.) of sulfathiazole every four hours. Three days after admission she was able to take a soft diet. A blood transfusion was given because the erythrocyte count fell to about 1,000,000. She continued to improve, but because of elevation of temperature, she was given a total of 965 grains (62.7 Gm.) of sulfathiazole over a period of twelve days, with an average of 80.4 grains (5.2 Gm.) daily. She was discharged fifteen days after admission in good condition, and follow-up observations revealed permanent healing.

In the second case the treatment consisted of warm moist dressings on the neck, mouth washes and the rectal administration of sulfathiazole, 120 grains (8 Gm.), the dose being repeated in twelve hours. Two days later the patient was given 15 grains (0.98 Gm.) of sulfathiazole every four hours by mouth; this was continued for eight days, after which all symptoms subsided, with full recovery. The patient was discharged from the hospital.

In the third case the general measures in the 2 preceding cases were employed, 30 cc. of a 5 per cent solution of azosulfamide being given intramuscularly in the first thirty-six hours, then 30 grains (1.95 Gm.) of sulfathiazole by rectum, after which 7½ grains (0.488 Gm.) of sulfathiazole was administered three times a day by mouth for the next eight days, and on the eleventh day after admission a transfusion of 300 cc. of citrated blood was given. Recovery was uneventful, and the patient was discharged fourteen days after admission.

The first 2 cases followed extraction of teeth; the third, a sore on the lip.

The author stresses that he does not advocate the exclusive routine use of the sulfanilamide drugs in treatment of this disease regardless of the stage or of the patient's condition when first seen. He admits that tracheotomy or wide incision or both may have to be resorted to, and, indeed, all such measures should be held in readiness at all times. He merely advocates the use of chemotherapy in cases in which respiratory and constitutional conditions are not so severe as to require precipitous surgical intervention and that conservatism is clearly indicated.

He concludes that "when a patient presents an inflammatory process involving the floor of the mouth, deep to the mylohyoid diaphragm, as evidenced by elevation of the tongue, and producing a brawny induration of one or both submaxillary regions, with or without involvement of the submental region, which is obviously not confined within the lymphatics but bounded by tissue planes" the diagnosis of Ludwig's angina is justified.

GORDON, Philadelphia.

Larynx

LARYNGEAL TUBERCULOSIS, WITH SOME OF THE MORE RECENT ADVANCES IN TREATMENT. FRANK R. SPENCER, Ann. Otol., Rhin. & Laryng. **50**:735 (Sept.) 1941.

Whether the larynx becomes infected by contact with sputum-laden bacilli or by metastasis is difficult to prove, even at autopsy. Investigations have shown that nearly all of the structures and organs of the upper respiratory tract contain a few tubercle bacilli even in persons who showed no clinical involvement of this area before death. Laryngeal tuberculosis is almost always chronic from the beginning, but acute and subacute exacerbations are not uncommon.

Hoarseness is the first symptom and may be present only in the morning or after coughing. It may be followed shortly by aphonia and difficulty in swallowing. Severe pain is a late symptom, but any pain indicates an unfavorable prognosis. Dyspnea appears if there is narrowing of the lumen of the larynx and may be serious if there is bilateral abductor paralysis. Objectively, the larynx may be pale, as most tuberculous patients are anemic, but it may be red if there is much cough. Infiltration of the interarytenoid sulcus is an early sign. If the infiltration spreads to involve the arytenoid cartilages these become enlarged. Ulcers may form. Edema is a late sign and means that the circulation has been interfered with. Unless fibrosis occurs the patient becomes progressively worse. Too much fibrosis may cause cicatricial stenosis. Perichondritis and chrondritis are usually rapidly fatal. A differential diagnosis between tuberculosis, syphilis and a malignant growth must be made.

In treatment, cauterization is the most important procedure. Vocal rest is vitally important. Heliotherapy, either with reflected sunlight or with ultraviolet radiation, may aid. Oil sprays and drops may be soothing, and use of 20 per cent chaulmoogra oil is advocated by some. To allay pain injections of alcohol into the superior laryngeal nerve may be made. Ionization, using special electrodes, may relieve pain. Chemotherapy has brought no striking results in controlling the lesions. Tracheotomy may be required to prevent asphyxia. Collapse therapy is believed to prevent laryngeal complications in many cases. If the lesion is confined to the epiglottis, amputation of this may prevent spread of the infection. Gastrostomy may be required to keep the patient with advanced laryngeal lesions from slow starvation, due to painful swallowing.

M. V. MILLER, Philadelphia.

LARYNGEAL TUBERCULOSIS: A STUDY OF 500 CASES OF PULMONARY TUBERCULOSIS WITH A RÉSUMÉ BASED ON TWENTY-EIGHT YEARS OF EXPERIENCE. JULIUS P. DWORETZKY and OTTO C. RISCH, Ann. Otol., Rhin. & Laryng. **50**:745 (Sept.) 1941.

The authors state that laryngeal tuberculosis is definitely decreasing, as a result of educational campaigns, improvement in examination, leading to earlier diagnosis and treatment of pulmonary lesions, and better treatment of tuberculosis, especially through increased use of collapse therapy. They classify all cases of pulmonary tuberculosis under the bronchiogenic, or bronchopulmonary, and the hematogenous type. The bronchiogenic type constitutes about 80 per cent of all cases; in this form the lungs are invaded by the bacilli through the air passages and the infection spreads to other parts of the lungs mainly by the bronchial route. In the hematogenous form the infection spreads through the lungs and other organs by way of the blood stream. After the disease is well established the type of infection is often mixed; a hematogenous lesion may ulcerate through the alveoli and become bronchiogenic, while a bronchiogenic lesion may ulcerate into the circulation and become hematogenous. Laryngeal tuberculosis is always secondary to pulmonary involvement and occurs from surface infection or by way of the blood stream, so that it is either bronchiogenic or hematogenous. Differential diagnosis is made on the following points:

Bronchiogenic Type	Hematogenous Type
1. Onset insidious, usually with pulmonary symptoms or fever, asthenia and loss of weight.	1. Onset acute, first symptoms usually laryngeal.
2. Chest signs of pulmonary involvement.	2. Chest signs few or absent.
3. Sputum often positive.	3. Sputum usually negative, later positive.
4. Characteristic roentgenographic signs of infiltration and often cavitation.	4. Roentgenograms show characteristic seeding.
5. First symptoms of laryngeal involvement usually hoarseness or irritation of larynx.	5. First symptoms usually dysphagia and sometimes aphonia.
6. Laryngeal involvement usually limited to a portion of larynx, most often posterior commissure.	6. Lesion usually diffuse and epiglottis invariably involved, often arytenoid cartilages and aryepiglottic folds.
7. Larynx usually involved alone and tonsils, pharynx, etc., rarely affected.	7. Tonsils, pharynx, soft palate and uvula often involved.
8. Prognosis usually good, especially if collapse therapy has been successfully applied.	8. Prognosis depends on character of infection. In cases of miliary involvement in which larynx is acutely involved, prognosis is hopeless and patient often dies in a few weeks or months, often with meningitis. If pulmonary infection is mild, laryngeal lesion often clears up without local therapy.
9. Usually requires little treatment and clears up with healing of lung.	9. Local treatment useless.

M. V. MILLER, Philadelphia.

Nose

THE PRESENT STATUS OF ROENTGEN THERAPY IN SINUSITIS. KARL KORNBLUM, Ann. Otol., Rhin. & Laryng. **50**:523 (June) 1941.

Kornblum presents a comprehensive review of the literature on roentgen treatment of sinusitis, which in many cases includes the technic used by the author in gaining his results. In view of the observations of other investigators and his

own results, he feels that roentgen therapy has passed beyond the experimental stage. He emphasizes that there must be close cooperation between the rhinologist and the roentgenologist and that there must be a proper selection of patients submitted to roentgen therapy. The selection must, of course, be made by the rhinologist. Most observers feel that a patient with acute sinusitis should not be subjected to irradiation; with this the author does not agree; he believes that, provided such a patient is under close observation of a competent rhinologist, he will as a rule obtain prompt relief. Subacute sinusitis probably reacts best to the therapy. Chronic hyperplastic sinusitis with exacerbations responds fairly satisfactorily, the results being in direct relation to the proportion of leukocytic infiltration and fibrosis of the sinal mucosa. Chronic sinusitis with extensive fibrosis may be treated with radiation and may sometimes show marked clinical improvement even though there is little demonstrable clinical or roentgenologic effect. If no improvement is noted after the first series of treatments, they should not be repeated. The presence of polyps and cysts does not contraindicate irradiation, and if these are composed of sensitive tissue they will respond. If the polyps are of the true fibrous type no results can be expected. Roentgen irradiation should not be used in cases of atrophic rhinitis. In treatment of sinusitis with asthma the results reported have been variable. The presence of asthma should not materially influence the roentgen therapy. Every victim of chronic sinusitis should be studied from the point of view of allergy, and, as the problem is a large one in many cases, treatment of the sinus situation alone will avail little. In the author's experience roentgen therapy has given satisfactory results in patients with vasomotor rhinitis. He also feels that the sinusitis of children presents an ideal field for this type of therapy and that if the treatment is carried out during childhood, many cases of chronic sinusitis will be avoided in adult life. He advises that the adenoids and the hyperplastic lymphoid tissue of the nose and throat be irradiated along with the sinuses. He discusses the technic and the reactions which may be expected.

M. V. MILLER, Philadelphia.

OZENA. M. M. CULLOM, J. A. M. A. **117**:987 (Sept. 20) 1941.

The author outlines several interesting features of this disease. He states: "Ozena is called also atrophic rhinitis, atrophic catarrh, coryza foetida, and sclerotic rhinitis. However, the favorite and common term is 'the opprobrium of medicine.' The Germans, with their genius for expressing delicate shades of meaning and their natural use of euphemistic terms, call it 'stink nose.' Because of the voluminousness of the literature, this review will be limited to the etiology."

Cullom then lists ten causes of ozena. He reviews in moderate detail some of the historical points in connection with the condition. Many first class authorities are quoted; some verbatim, others by transliteration.

He reaches certain conclusions based on observation, experience and collaboration. He feels that ozena is the end result of a chronic purulent focus, which focus may exist in the nasal cavity, the accessory sinuses or the postnasal space. This conviction is likewise expressed by Grünwald and, to a lesser extent, by Michel, although Löwenberg is the stoutest defender of the sinusitis theory.

The bedrock of the author's conviction is the brilliant observation of Bosworth that there is a purulent rhinitis of early childhood which results in ozena in from five to ten years.

Ozena appears five times as often among females as males. The reason advanced for this is that boys are essentially outdoor animals, and through participation in games and activities that call for exertion and quickened circulation, with resultant improved nutrition, improved states of the turbinal venous sinuses take place, whereas girls are indoors, in dry atmospheres, and do not partake of vigorous physical activity, with resultant lesser nutritional benefits, and so often are apt to acquire, or become a prey to, a deficiency disease.

Several more interesting points along this line are developed, but it would be difficult to follow them further without lengthening this abstract unduly.

In discussion, Dr. Joseph C. Beck stated that the thoroughness with which the present group of pediatricians and pathologists have studied certain phases of this problem may account for the probability that one sees fewer cases of atrophic rhinitis now than formerly. In his opinion, disease of the sinuses plays an important role in the causation of atrophic rhinitis, but there may be some other underlying factor that has not thus far been discovered. The best method of dealing with atrophic rhinitis is local and conservative, not radical or surgical, treatment of the sinuses. Roentgen examinations reveal the fact that children who have clear sinuses show better pneumatization, and Shea, Carmody and many others have contributed papers showing that sinal disease may prevent the free aeration and development of the sinuses.

GORDON, Philadelphia.

THE TRANSANTRAL APPROACH TO THE ETHMOIDAL AND SPHENOIDAL SINUSES. J. BOWRING HORGAN, J. Laryng. & Otol. **16**:290 (Aug.) 1941.

Fifteen years ago the author first published his observations on the transantral approach to the ethmoid and sphenoid sinuses. Since that time considerable interest has developed in this approach. This contribution deals with modifications of the original technic rather than with a minute discussion of the anatomic and physiologic considerations, as previously described.

With regard to operations designed to cure infection of a sinus by the creation of a large opening for drainage, it is postulated that this procedure may be sufficient in the early stage, but once polypoid degeneration has taken place, recovery is not likely.

The inferior turbinate should never be removed, even partially. The middle turbinate may require removal, either whole or in part, but should be conserved when possible. When it can be retained it is fractured outward into the empty ethmoid "box" at the completion of the operation.

The transantral approach, contrary to popular belief, is not necessarily designed for cases of ethmoiditis in which there is also involvement of the mucosa of the antrum: It is suitable for those cases in which there is little or no disease in this sinus.

The facility with which sphenoethmoid drainage can be effected depends on the fact that the lamina papyracea and the roof of the antrum meet at an angle opening upward and outward. This explains why it is so simple for an instrument working across the antrum to break into the comparatively large, and often self-evident, posterior ethmoid cells.

A short oblique incision is considered superior to the usual horizontal one, and free removal of the canine face of the maxilla, roughly in the shape of a "V," is recommended.

If the sphenoethmoid opening is satisfactorily performed, there is little likelihood of stenosis. Care should be exercised to avoid injury to the orbital periosteum or the dura. Details of the toilet of the wound and the postoperative management and a discussion of the choice of anesthesia complete the presentation.

LEJEUNE and BAYON, New Orleans.

Miscellaneous

PNEUMOCOCCIC MENINGITIS: CASE REPORT OF COMPLETE RECOVERY IN A TEN MONTHS OLD INFANT. ARTHUR J. MOSS and EDWARD L. STREM, Arch. Pediat. **58**:203 (April) 1941.

A white girl 10 months of age recovered from type 14 pneumococcus meningitis after the use of specific rabbit serum and sulfapyridine. The first spinal fluid culture, taken before the onset of treatment, was positive for the type 14 pneumococcus. Subsequent cultures of the spinal fluid, taken after treatment was initiated,

were all sterile. The level of sulfapyridine in the blood was maintained between 23 and 26 mg. per hundred cubic centimeters, while the concentration of the drug in the spinal fluid was between 16 and 18 mg.

ORR, Buffalo [AM. J. DIS. CHILD.].

CEREBRAL COMPLICATIONS FOLLOWING SURGICAL OPERATIONS: II. FACTORS WHICH PREDISPOSE TO CEREBRAL ANOXIA. ALBERT BEHREND and HELENA E. RIGGS, Arch. Surg. **41**:772 (Sept.) 1940.

Behrend and Riggs have demonstrated that most types of postoperative cerebral complications may occur as a result of cerebral anoxia. In the cases studied anoxia was usually secondary to an acute general circulatory collapse precipitated by administration of an anesthetic plus the trauma of operation in patients whose margin of circulatory reserve had been reduced. In many cases the presence of such a preexisting chronic circulatory insufficiency was unsuspected either by the patient or by his physician, but histologic observations in the vital organs were interpreted as evidence of such a disturbance. While the cerebral changes appeared to be the result of anoxia operating through general circulatory insufficiency, it was evident that such anoxia could be produced by various mechanisms and that more than one mechanism could be present in a given case. There are four main types of anoxia. The first is stagnant anoxia, in which, because of slowing of the blood current, tissue asphyxia occurs. The second is anemic anoxia, which occurs when the hemoglobin content of the blood is too low to allow sufficient oxygen to be carried to the tissues. The third is histotoxic anoxia, which occurs when the cells are so damaged as to be unable to utilize oxygen. A common cause of this condition is the overenthusiastic use of morphine, barbiturates, anesthetic drugs or alcohol. The fourth is anoxic anoxia, which may occur throughout the body as a result of an insufficiency of the available supply of inspired oxygen or locally as the sequel of trauma, acute inflammation or gas gangrene. Among the factors important in determining a predisposition toward anoxia is organic heart disease. In 76 per cent of the cases studied post mortem by the authors an organic cardiocirculatory lesion existed prior to operation. Other factors listed as predisposing causes are anemia, age, metabolic disorders, vitamin deficiency, surgical emergencies, such as shock, hemorrhage and overwhelming infection, and the type of anesthesia used.

GRANT, Philadelphia [ARCH. NEUROL. & PSYCHIAT.].

DAILY VARIATIONS IN THE BREATHING CAPACITY AND GRIP STRENGTH OF PRESCHOOL CHILDREN AS RELATED TO THE OCCURRENCE OF COLDS. ELEANOR METHENY, Child Development **12**:69 (March) 1941.

The relation of day to day variations in the breathing capacity and grip strength to the occurrence of colds was investigated. The subjects, 22 children 4 years old, were tested daily for two periods of four weeks and six weeks, respectively. Decreases in grip strength and in breathing capacity were found to occur more frequently on the days prior to the occurrence of a cold than on days not followed by a cold. Criteria were set up for the "prediction" of colds one or two days previous to the actual occurrence of the cold. Tetrachoric correlations between predictions of "cold" or "no cold" and the occurence of non-occurrence of a cold ranged from 0.54 to 0.81, the actual figure depending on the criterion used.

AUTHOR'S SUMMARY [AM. J. DIS. CHILD.].

CEREBROSPINAL RHINORRHEA: PATHOLOGIC OSBSERVATIONS. MAX L. SOM and RUDOLPH KRAMER, Laryngoscope **50**:1167 (Dec.) 1940.

Som and Kramer report the pathologic observations in 2 cases of cerebrospinal rhinorrhea and subsequent death from meningitis. One patient had severe headaches, for two years relieved only by lumbar punctures and then for three years

relieved by the escape of large amounts of clear, watery fluid through the nose. During two weeks of hospital care she had hypertension and purulent meningitis, with the influenza bacillus present in the spinal fluid. Roentgenograms indicated an intrasellar tumor. Autopsy showed diffuse suppurative meningitis and a large neoplasm of the pituitary. The paranasal sinuses were removed in block and cut in serial sections. Microscopic examination showed a fistula extending from the floor of the anterior fossa to the olfactory recess. A polyp, covered by an extension of dura and with a core of nerve tissue, occurred at the nasal opening of the fistula. The fistula in the cribriform plate led to an opening in the dura and was surrounded by an area of osteomyelitis.

The second patient was admitted to the hospital with pneumococcic meningitis. Intranasal examination showed acute bilateral sphenoethmoiditis with polypoid changes. The left sphenoid and ethmoid sinuses were exenterated. The following day there was a flow of cerebrospinal fluid from the opposite, intact side. Autopsy three days later showed diffuse purulent meningitis and a defect in the dura 1 cm. in diameter on the cranial surface of the right, intact sphenoid sinus. Microscopic sections of the sinuses showed a submucous abscess in the posterior wall of the right sphenoid sinus, with adjacent osteomyelitis and erosion through the bone to the defect in the dura.

WOOD, Newark, N. J.

REUNION OF STUMPS OF SMALL NERVES BY TUBULATION INSTEAD OF SUTURE. PAUL WEISS, Science **93**:67 (Jan. 17) 1941.

Approximation of the cut ends of a nerve by suturing can never be precise enough to prevent masses of fibers from escaping into the surroundings and straying off to uncontrollable destinations. The difficulty can be met by tubulating the nerve ends with a tightly fitting cuff of fresh artery.

For instruments two pairs of hard steel (watchmaker's) forceps, ground down until the ends have become very slender and sharp pointed, are used to manipulate the artery and the nerve. Under Ringer's solution one pair of forceps is used to hold the end of the artery dilated, while the other grasps the edge of the nerve and introduces it into the arterial cuff. The cuff provides a firm link between the nerve ends and prevents the formation of a neuroma and the uncontrollable escape of fibers. It is gradually transformed into perineural connective tissue and after several months can no longer be identified. Regenerating fibers traverse the wound in parallel courses, instead of in the usual confusion following nerve suture.

H. A. MILLER, Philadelphia.

HEAD INJURIES AND MENINGITIS. E. A. LINELL and W. L. ROBINSON, J. Neurol. & Psychiat. **4**:23 (Jan.) 1941.

Linell and Robinson report 7 cases of pneumococcic meningitis following fracture of the base of the skull. In the first 4 cases the meningitis developed rapidly —in case 1 it followed a simple fracture of the nasal bones and in case 2 a basal fracture of the skull opening into the various sinuses, and in cases 3 and 4 the infection spread along the line of fracture from a focus in the middle ear. In cases 5, 6 and 7 the meningitis occurred five, fourteen and two and a half years, respectively, after rather trivial injuries, which, however, provided pathways for the subsequent infection.

MALAMUD, Ann Arbor, Mich. [ARCH. NEUROL. & PSYCHIAT.]

CEREBRAL ABSCESS DUE TO PFEIFFER'S BACILLUS—SIX YEARS AFTER PFEIFFER'S BACILLUS MENINGITIS. T. G. I. JAMES, Proc. Roy Soc. Med. **33**:628 (Aug.) 1940.

A child aged 9 years was admitted to the hospital in a lethargic state, with rigidity of the neck, a positive Kernig sign, right hemiplegia and slight papilledema. At the age of 2 years the child had suffered from influenzal meningitis, and

Pfeiffer's bacillus was isolated from the cerebrospinal fluid. After recovery the child still had right hemiplegia. Lumbar puncture on this second admission yielded a clear fluid greatly increased under pressure, which contained 26 cells per cubic millimeter. A roentgenogram of the skull showed a calcified lesion in the left frontoparietal area. The patient became increasingly lethargic and a month later was unconscious. The left frontoparietal region was explored through a burr hole; a calcified area was found under the dura mater, and a large abscess was opened and 3 ounces (89 cc.) of pus obtained. Culture of the pus yielded Pfeiffer's bacillus. The child regained consciousness. Further treatment consisted in repeated aspirations of the abscess. The wound finally healed, and the child was free of symptoms apart from the right hemiplegia, which remained unchanged.

WILLIAMSON, New Orleans [AM. J. DIS. CHILD.].

NEUROLOGIC SYMPTOMS OF UVEOPAROTID FEVER. P. OTTONELLO and T. ANARDI, Riv. di pat. nerv. **56**:185 (Sept.-Oct.) 1940.

Ottonello and Anardi report the case of a woman aged 58 who, without any prodromal symptoms, exhibited total paralysis of the left facial nerve. This was followed in two days by transient slight bilateral swelling of the parotid gland. Total paralysis of the right facial nerve and disturbance of deglutition and function of the vocal cords appeared one week later. Inflammation of the parotid gland became manifest one month later. Anisocoria and pupillary rigidity appeared in forty-five days and acute polyneuritis of all four extremities in seventy-five days. The deep reflexes disappeared in the course of the disease, and signs of involvement of the cardiac fibers of the vagus nerve became evident. The lymph nodes were not enlarged, with the exception of the hilar nodes, which showed moderate enlargement. All the symptoms except the left facial paralysis disappeared within five months. The tuberculin cutaneous test and the examination of the sputum for tubercle bacilli gave negative results. The cerebrospinal fluid exhibited changes of a meningeal character, slight lymphocytosis and slight increase in the globulins. The diagnosis of uveoparotid fever was verified by biopsy. The authors conclude that paralysis of the facial nerve and neurologic complications of the uveoparotid fever are caused by selective localization of the unknown virus of the disease in the nerves.

J. A. M. A.

THE SURGICAL TREATMENT OF THE NERVOUS SYSTEM. WILLIAM SHARPE, Arch. venezol. Soc. de oto-rino-laring., oftal., neurol. **1**:77 (Sept.) 1940.

In reviewing his neurosurgical experience for the past twenty-five years, Sharpe has classified his results by reference to three groups of pathologic conditions:

1. Conditions in which neurosurgery is limited in value, as (*a*) tumor of the brain, (*b*) abscess of the brain, (*c*) internal hydrocephalus and (*d*) epilepsy.

2. Conditions in which excellent results have been obtained, as (*a*) trigeminal neuralgia, (*b*) lesions of the medulla, (*c*) lesions of the peripheral nerves, (*d*) external hydrocephalus, (*e*) recent trauma of the brain in adults, in children and in the newborn.

3. Chronic traumatism of the brain, including (*a*) post-traumatic "wet" brain and (*b*) post-traumatic neurosis.

Group 1.—(*a*) In speaking of tumor of the brain, the author states that if the tumor is diagnosed early, is small and does not, either by encroachment or by infiltration, destroy too much of the vital intracranial structures, its extirpation is possible, and the patient may benefit sufficiently to return to a more or less normal physical and economic state. The author believes that while ventriculography is important as an aid in diagnosis, encephalography is definitely more valuable.

Tumors of the motor area or at the cerebellopontile angle, fibroma of the eighth nerve and tumors that extend to the meninges are usually malignant. Meningeal endothelioma is benign and not of a true cerebral neoplastic nature. In this general group the author had an operative mortality of 18 per cent.

(*b*) In cases of cerebral abscess in which the brain was not operated on, there was a mortality of 100 per cent. However, depending on the underlying pathologic condition, on the time of operation and on the size and the location of the abscess, the operative mortality varied from 24 to 48 per cent. The author favors subtemporal decompression and a "clean" approach to the abscess cavity.

(*c*) Internal hydrocephalus is usually produced by a block somewhere in the cerebrospinal drainage system. A frequent cause is a subcortical hemorrhage. There is a malignant form, the symptoms of which are rapidly progressive and the prognosis grave, and a benign form which can be diminished by frequent lumbar punctures.

(*d*) Epilepsy does not yield to any form of neurosurgical treatment.

Group 2.—The author's most favorable results were obtained in this group: (*a*) In trigeminal neuralgia, section of the posterior sensory roots or even extirpation of the ganglion produced the best results. On the other hand, section of the second and third branches of the nerve failed in 25 per cent of his cases.

(*b*) Among lesions of the medulla is primary tumor of the medulla. This was malignant in 44 per cent of the cases, but a correct diagnosis could be made readily because of the early appearance of localizing symptoms. The immediate operative mortality was 8 per cent. However, there were frequent recurrences, and most of the patients succumbed within five years. Traumatic lesions of the medulla are associated with fractures of the cervical vertebrae, and the prognosis depends on the extent of the destruction of the underlying tissue. If considerable laceration of the spinal cord is present, the results will be poor. The author's operative mortality in cases of acute trauma was 13 per cent.

(*c*) With injuries to the peripheral nerves in general, early anastomosis of the severed ends usually brings excellent results. For injury of the brachial plexus during childbirth the author advocates repair sometime between the third month and the first year of the child's life. However, perfect restoration of function in the affected arm is rarely achieved. For peripheral facial paralysis he either makes an anastomosis with the twelfth (hypoglossal) nerve or approximates the severed ends of the seventh nerve. When infection has been a factor, as in otitis or mastoid disease, he may merely expose the nerve and free it of any adhesions or granulations.

(*d*) His results in the treatment of external hydrocephalus were affected by the underlying pathologic condition. If the basic lesion was diffuse meningitis, meningoencephalitis or profuse supracortical hemorrhage, the prognosis was grave. On the other hand, when there was either no or only slight damage to the cortex, frequent lumbar punctures usually produced a cure. His mortality rate was 6 per cent.

(*e*) In dealing with recent cerebral injuries in adults, the essentials of treatment depend on absolute rest and the control of shock. When there is evidence of increased intracranial pressure, the author employs frequent lumbar punctures, alone or augmented by either a magnesium sulfate or a sodium chloride regimen. Operative intervention is contraindicated during the initial period of shock and in the terminal state, in which it will only hasten death. The treatment of cerebral injuries in children, especially intracranial hemorrhages of the newborn, is routinely frequent spinal drainage. If there is severe and persistent increase of intracranial pressure, the author often supplements this with subtemporal decompression and cisternal puncture. His results from employing spinal drainage as the ideal method of treatment show a 34 per cent mortality.

Group 3.—In some cases of chronic traumatism of the brain due to an old fracture there is a greater or lesser degree of wetness of the brain, which is

responsible for the symptoms. In other cases a post-traumatic neurosis develops, which may be either functional or organic. Careful clinical examination plus lumbar puncture often helps in establishing a correct diagnosis. The lumbar puncture frequently produces a cure in cases in which there is an increase of intracranial pressure. Should spastic paralysis of any type occur, spinal drainage must be supplemented with orthopedic treatment.

In conclusion, Sharpe advises against procrastination in dealing with cerebral injuries since the best results are obtained by spinal drainage when the subcortical hemorrhage is still liquid.

PERSKY, Philadelphia.

THERAPEUTIC USES OF MAGNESIUM SULFATE COMBINED WITH EPHEDRINE. ALFREDO VIDAL FREYRE, Rev. med. y cien. afines **12**:938 (Dec. 30) 1940.

Including in the present report some of his previous observations on the use of hypodermic injections of magnesium sulfate and ephedrine, the author surveys his results in the application of this treatment to a number of correlated conditions.

He reports either cure or improvement in a series of cases of bronchial asthma, nasal asthma, infantile asthma, urticaria and serum reactions, eczema and similar allergic states. He feels that since the magnesium sulfate exerts an antispasmodic, sedative, desensitizing, hypnotic and moderately hypotensive function, its augumentation by ephedrine hydrochloride supplies an additional antispasmodic, antiallergic and bronchodilator action. He has found this treatment particularly valuable with many patients who have become "epinephrine fast."

In most of his cases one injection is sufficient to either cure or relieve the acute symptoms. Sometimes it is necessary to repeat the injection the following day. The preparation which he uses contains 0.05 Gm. ephedrine hydrochloride, 1.25 Gm. magnesium sulfate and distilled water up to 5 cc. For adults the dose is 5 cc. of this solution and for children 3 cc. However, for children the amount of ephedrine hydrochloride is reduced to 0.015 Gm.

PERSKY, Philadelphia.

LYMPHOCYTIC CHORIOMENINGITIS. CARLOS ALBERTO VIDELA and JOSÉ L. FARINA, Rev. med. y cien. afines **12**:961 (Dec. 30) 1940.

The authors present 2 proved cases of lymphocytic choriomeningitis. In addition, they review the recent literature on the subject, especially from the standpoint of symptomatology. They proved their cases by intrathecal inoculation of white rats. Twelve days later pathologic examination of the brains showed exudative meningitis, involving particularly the choroid plexus and the ventricles. Detailed microscopic studies of the brain, however, are not included in this report.

PERSKY, Philadelphia.

SYMPTOMATOLOGY OF STUTTERING. ALFREDO DUB, Rev. méd. latino-am. **25**:1135 (July) 1940.

The cause of stuttering may be systemic or psychic. With stuttering of systemic, or somatic, origin there is a disturbance of respiration. The abdominal and the thoracic muscles function independently and not synchronously as they do in normal breathing. The involuntary muscular movements which accompany the difficulty in breathing are spasmodic; they may be either tonic or clonic. The facial muscles are involved, so that the patient uses various gestures, and unintentional movements occur, as he strives, to express himself. It is difficult to differentiate between symptoms of systemic and symptoms of psychic origin, because they are closely related. For example, a person may not stutter in one environment, while in another he can be understood only with difficulty. Respiratory curves showing alterations observed in cases of stuttering illustrate the article.

DWYER, Kansas City, Mo. [AM. J. DIS. CHILD.].

REMARKS ON F. J. MAYER'S PUBLISHED WORK "DACRYOCYSTORHINOSTOMY BY WAY OF THE APERTURA PIRIFORMIS." K. KOFLER, Monatschr. f. Ohrenh. **74**: 299 (June) 1940.

In defending his own type of surgical approach to the tear sac, Kofler says that the amount of bone to be removed in his operation is only half that which is necessary in Marschik's procedure. The transeptal approach of Kofler is not disadvantageous, as the septal incision heals well and the lateral nasal wall is more readily visualized. In Marschik's type of operation the nasolabial fold may be disfigured. After-treatment in Kofler's operation is simple, and in 220 cases Kofler has seen only one small cutaneous abscess. Otherwise his results have been uniformly excellent.

LEDERER, Chicago.

REPLY TO KOFLER. F. J. MAYER, Monatschr. f. Ohren h. **74**:306 (June) 1940.

Mayer defends his position that it is the duty of every surgeon to report the results of a new method. Marachik's method did not have any bad cosmetic effects, nor was the maxillary sinus opened involuntarily. The method allows for more thorough exenteration of the surrounding ethmoid cells and gives better access to the tear sac. It seems to Mayer that this approach fulfils the same intention that Kofler has in the transseptal procedure.

LEDERER, Chicago.

ARTICULATION OF DEFINITE LETTERS IN THE GREEK LANGUAGE IN ROENTGEN PICTURE. J. CHRYSIKOS and G. E. JANNULIS, Monatschr. f. Ohrenh. **74**:407 Aug.) 1940.

Roentgen studies of various letters of the Greek alphabet were undertaken and the position of the tongue made visible by a slight coating of barium sulfate.

LEDERER, Chicago.

SULFANILAMIDE TREATMENT OF TWENTY-TWO PATIENTS WITH MENINGOCOCCIC MENINGITIS AND SULFANILAMIDE AND SULFAPYRIDINE THERAPY OF FOUR PATIENTS WITH PNEUMOCOCCIC MENINGITIS. A. J. R. E. VAN SCHOONHOVEN VAN BEURDEN and T. LOONSTRA, Nederl. tijdschr. v. geneesk. **84**:3673 (Sept. 21) 1940.

Of the 19 patients with meningococcic meningitis treated by the authors from 1932 to 1938, 15 died. After the introduction of sulfanilamide treatment the mortality rate fell considerably, especially in very young patients. Of 8 patients 2 years old or less observed by the authors, only 2 died; of 8 adults, no less than 4. Although the doses of the sulfanilamide compounds given these patients were only moderate, the writers are led to assume, from facts observed by them and from comparative considerations, that it is improbable that the fate of patients with meningococcic meningitis is solely determined by the amount of sulfanilamide given, so that a certain dosage does not warrant a success of this therapy. The prognosis of pneumococcic meningitis, even after the introduction of sulfanilamide treatment, remains a dubious one; of the 4 patients, 3 died. The result of energetic treatment was a rather moderate one.

VAN CREVELD, Amsterdam, Netherlands [AM. J. DIS. CHILD.].

Society Transactions

AMERICAN OTOLOGICAL SOCIETY, INC.

ERNEST M. SEYDELL, M.D., *President*

ABSTRACTED BY ALFRED LEWY, M.D.

Seventy-Fifth Annual Meeting, Atlantic City, N. J., May 28-29, 1942

President's Address: The Early Development of the Teaching of Otology in Europe and America. DR. ERNEST M. SEYDELL, Wichita, Kan.

On July 22, 1868, seventeen members of the American Ophthalmological Society organized the American Otological Society. One hundred years ago otology did not have a respectable standing. John Abernathy at the height of his fame refused to sanction the opening of a hospital for patients with diseases of the eyes and ears in London. Thirty-two years ago the otolaryngologist was just another spray bottle doctor.

The first record of the introduction of otology was made in 1861 at the Allgemeinen Krankenhaus in Vienna. By 1878 fourteen German universities had courses and clinics in otology, which by 1911 were made obligatory.

Instruction in otology was first given at Dartmouth Medical School in 1824, at Jefferson Medical College in 1827, at the medical school of Columbia University in 1841, at that of the University of Maryland in 1867, at Harvard Medical School in 1869, at the medical school of Yale University in 1876 and at that of the University of South Carolina in 1881. Columbia University made it obligatory in 1870, the University of Pennsylvania in 1874, the University of Vermont in 1876, Yale University in 1878 and the University of South Carolina in 1881. The first otologic clinic was established by Elisha North, in New London, Conn., in 1817. The New York Eye Infirmary was established in 1820; its affiliate title was changed to Eye and Ear in 1864.

On the fiftieth anniversary of the American Otological Society the members entered the first World War and on the seventy-fifth they again find themselves engaged in a world conflict. If the peace to follow is to be lasting, all must give of themselves to their country in peace as well as in war. Many of the younger members are already in service, others are teaching the future medical officers, while to those unable to be of active service is left the care of the health of the defense workers and the civilian population. Despite the burden, otologists must help maintain scientific medicine. Valuable contributions can be made by those in the armed service.

The members of this society must also be guardians of American aims and principles.

I wish to express my deep appreciation of the honor of serving as president of this society during the past year.

Otology and Aviation. DR. RALPH A. FENTON, Portland, Ore.

The Medical Research Board was established by the United States Army through Col. Theodore C. Lyster and Col. Eugene Lewis in 1917 for the investigation of mental and physical factors pertaining to military aviation. It was headed by Prof. Yandell Henderson, with the collaboration of the late Dr. John B. Rae. The first commandant of the Air Medical School was Colonel Louis H. Bauer.

The early emphasis on labyrinthine function has been replaced by investigation of vision and the ocular muscle balance, depth perception and the neuropsychic, cardiovascular and respiratory functions in the light of experimental evidence and of flight conditions in the stratosphere. The auditory apparatus, especially the eustachian tube, overshadows the labyrinth in importance. The flyer working from instruments must learn to disregard the lagging impulses from his labyrinthine canals and follow the instruments.

Any disease of the external auditory canal or abnormality of the tympanic membrane except small, well healed scars not impairing mobility, and evidence of a radical mastoidectomy disquality; likewise, a not well healed site of a simple mastoidectomy. A single attack of otitis media without recurrence does not disqualify. Repeated attacks and recurrent tinnitus, especially if there are other findings, do.

The whisper test is used, and a grade of 20/20 is required for the original entrance into the Air Corps, but experienced aviators on reexamination are permitted 8/20 or 15/20 in one and 5/20 in the other ear. Audiometric tests are recorded annually when possible. On the original examination the hearing loss must not exceed 15 per cent. The patency of the eustachian tube is tested, and blocking disqualifies. Equilibrium tests are simplified. Ordinarily the applicant stands on one bare foot and flexes the opposite knee to a right angle without bending the hip and holds it for fifteen seconds. He repeats this, standing on the other foot. The turning test is made if the history suggests it. The caloric test is done with a syringe only if suggested by the history or other findings. The radio beam is not found to cause fatigue when controlled by a rheostat. Noise and vibration are somewhat controlled by the helmet and the radio head set. The other personnel may plug their ears. Sound proofing of cabin type planes eliminates much of the noise. The loss of hearing for the higher frequencies in older pilots is not significantly greater than that in civilians of similar age. Apparently, the loss around 4096 double vibrations is due to greater vulnerability of the basal coil.

Changes in air pressure interfere with the lower frequencies. In ascent the pressure within the tympanum becomes greater than that outside. The tube opens or may be helped to open by swallowing or yawning. During descent at more than 300 feet per second the tube is opened with greater difficulty; increased pressure in the nasopharynx tends to keep the tubes closed and may cause pain, local congestion and transudate or hemorrhage in the middle ear with deafness for low tones, the condition known as acute aero-otitis media. Repeated episodes of this type may lead to chronic changes. Flying should be prohibited for those suffering from rhinopharyngeal infections.

Sudden descent drives blood to the head, increasing intracranial pressure, and may produce minute hemorrhages; sudden rise produces ischemia, followed by active hyperemia and eventually the same effects. Intratympanic pressure plus anoxemia may cause fainting in rapid ascent to 15,000 or 20,000 feet (4,570.5 or 6,475 meters). The oxygen mask and the sealed low pressure cabin obviate this. Airsickness, like seasickness, is overcome by training. Oversensitive persons, who do not adjust, usually show other signs of instability.

Gravity and motion are perceived through muscular, articular, visceral and cutaneous sensations as well as through vision and vestibular reactions. Faulty interpretation must be trained out, especially in "blind" or instrument flying. Phlegmatic pilots with relatively insensitive labyrinthins do best. The "feel of the plane" is important. The pilot must acquire confidence in his instruments and disregard his sensations. Excessive labyrinthine sensations are hazardous.

The toxic action of tobacco seems harmful only to susceptible persons. Alcohol may produce recklessness. It also produces anoxia of the tissues and lowers the flyer's ceiling. Worry predisposes to circulatory upsets. A feeling of exhilaration or weakness at high altitudes calls for oxygen. Mucosal vasoconstrictors must be used carefully, as they lead to secondary engorgement. Persons with

high blood pressure or impaired coronary circulation undergo distinct hazards. Embolism caused by nitrogen bubbles is not considered a factor in unconsciousness in substratosphere flyers. The civilian flying below 15,000 feet is not liable to decompressive hazards.

Flight surgeons are doing an excellent job, and their standards are practical. This is attested by the excellent physical condition of civilian pilots over a period of years. The younger pilots do not lose hearing as did the pioneers.

Otologists should acknowledge their debt to the pioneer flight surgeons, notably Bauer, Tuttle, Armstrong, Campbell and Hargreaves. Colonel Tuttle and Major Campbell aided in the preparation of this review.

DISCUSSION

DR. CHARLES T. PORTER, Boston: The vestibular examination is regarded as of much less importance in this than in the last war. The eustachian tube, with its mechanics, and the middle ear are rightly much more emphasized. The ocular examination has been greatly increased, especially with respect to depth perception and muscle balance, and more attention is given to the psychologic as well as to the general physical examination. The flight surgeon has taken over the job, and the otologist is merely an accessory.

Dr. Harry G. Armstrong, director of the Aero Medical Research Laboratory, supplied my historical data. The first to note changes due to altitude were Glaisher and Coxwell, who ascended to 29,000 feet (8,839 meters) in a balloon in 1862. They gave an accurate account of the effects of anoxia, such as loss of visual acuity and of hearing, paralysis and finally unconsciousness. This aroused no medical curiosity. The first medical investigation of aviation was reported by a Frenchman, Paul Bert, and his student, Tissandier. The latter with two other men made a flight up to 28,800 feet (8,779 meters). His two companions died, but Tissandier recovered and reported his experience to Bert.

Several men tried unsuccessfully various methods of flying with helicopters and with steam-driven and spring-driven airplanes. Cayley, Henson and Stringfellow in England, Lebris and Chanute in France and Lilienthal in Germany gave some impetus to this, but the impetus to practical aviation came with the Wright Brothers' first successful flight in 1903 at Kittyhawk, N. C.

In 1910 Germany drew up the first set of standards for military pilots and in 1915 had a functioning service for medical control of aviation; France, in 1917. In this country the army contracted for its first airplane in 1908 and had its first fatality, the death of Lieut. Thomas Selfridge, in September of that year. The first instructions for the examination of candidates for aviation duty were published in this country on Feb. 2, 1912. The first candidates to take and pass the Army examination were Lieuts. F. J. Gerstner and F. T. Armstrong.

In 1917 Major (later General) Theodore C. Lyster and Lieut. William H. Wilmer revised the Army schedule of examination, and a month later Col. I. H. Jones drew up an entirely new examination form.

In October 1917 a mission was sent abroad to study aviation and aviation medicine. In the meantime a medical research board and laboratory were established in this country at Hazelhurst Field. In May 1919 the School of Flight Surgeons was established and, along with a research laboratory, was moved to Mitchell Field, L. I. In 1922 the name was changed to the School of Aviation Medicine and moved to Texas.

After the war, interest died down but was revived by the work of Major D. A. Myers on the physiology of blind flight about 1931 to 1933. Lieut. Col. McGrow in 1933 demonstrated the need for an aero medical research laboratory, and authority to establish it was given in May 1935, and it was functioning by January 1937. The Civil Aeronautics Society established a similar laboratory in Kansas City in 1938, under Dr. Wade H. Miller, and from that time on medicine came into its own in aviation, but from the early twenties to the early thirties aviation medicine was practically at a standstill in this country.

Dr. Arthur W. Proetz, St. Louis: The relatively recent advent of aviation together with war, which has brought it home to everybody, teaches one that one has to keep abreast of scientific advance in all its branches.

Not only doctors but the human mechanism keeps up with it. When railroads were introduced, it was predicted that the human nervous system could not long stand the speed of fifteen miles (24 kilometers) per hour. When the automobiles came in, a new type of person, the chauffeur, cleancut, tense, with a well developed nervous system, appeared. Now that a grandmother drives better than he did, the type has reappeared in the aviator. You see the pilot as a well trained, alert type, with his voluntary and involuntary senses adapted to his job.

The second lesson one learns from Dr. Fenton is that in most situations elaborate scientific tests can be replaced by more practical ones as is done in the Army. The lessened importance of the vestibular tests alone exemplifies this trend.

In regard to smoking habits, one man may overdo with two cigarets while another man may smoke all day without apparent harm.

The solution I mentioned before the American Laryngological Association may prove useful in application to the examination of the eustachian tubes of stratosphere fliers, as it appears to have no effect on the circulation and the heart action.

Dr. Harris P. Mosher, Boston: When aviation medicine became prominent, it was the heyday of vestibular tests. These tests have sunk and now, understandably, occupy a minor place. The humble eustachian tube is more prominent.

Dr. Bunch was of the opinion that some of the aviation crashes were probably due to the aviator's not being able to hear his beam. The trauma of flying had dulled his hearing. Has the protection given by the modern plane and the aviator's helmet done away with that hazard?

Dr. Edmund P. Fowler, New York: Thirty-five years ago my associates and I improvised this little balloon and have been using it ever since with children, as supplementary treatment. I am glad the balloon has been born again.

Dr. E. R. Lewis, Los Angeles: May I add just one word? I want to put this on record. Dr. Paul Halloran in 1912 headed a commission appointed by the Surgeon General and established the first 609. That was reviewed in the spring of 1917, and his name and that of Dr. Lyster should be on the record as being largely responsible for the first codification of the requirements for the Army in flying. Dr. Wilmer's first contact with the flying service was in August 1917, when he was appointed the ophthalmologic head of the Medical Research Board.

Dr. Ralph A. Fenton, Portland, Ore.: I was going to acknowledge Colonel Tuttle's debt to Dr. Fowler's inventive genius. This thing in the hands of the nurses, who teach their passengers to use it, is a useful adjunct to their own efforts to get their heads opened up by yawning and stretching their necks. I am grateful to Dr. Porter for going into the early history, which included the work of many of the members of this society, and to Dr. Proetz for speaking of the physiologic effect of the solution which he mentioned. Paul Bert was honored by the French aviation group in the creation of a magnificent laboratory at LeBourget field.

The Army has set standards to prevent waste of life and material, and the psychologic examination is more important than the otologic, although they dovetail. The aviator must be of the type of Shaw's hero in "Man and Superman."

Regarding Dr. Mosher's keen analysis, there has been much discussion of the accidents happening around Salt Lake, Utah. Failure to hear the radio beam has been eliminated as a cause by the examiners of the United States Department of Commerce. During landing, two pilots are listening in. They used to just edge in, skimming too close to the Wasatch Mountains, which are 6,000 feet high (1,828.5

meters). They now come in at 15,000 feet and take half an hour to land, and have had little trouble lately. The loudness of the beam is controlled by a rheostat under the finger of the pilot. In my judgment, and I have flown over that route a great deal, the hazard has been eliminated.

Mycosis of the Middle Ear and Mastoid. DR. JOHN J. SHEA, Memphis Tenn.

Mycotic infections of the middle ear and mastoid process are more frequent than the literature would indicate. Only casual references are made to its extension from the external auditory canal to the middle ear. Multiple perforations occur more frequently than in tuberculosis. Its diagnosis is made positive by (1) the clinical picture, (2) the type of fungus isolated, (3) the response to fungicidal agents and (4) the cutaneous allergic and serologic reactions.

Fungous infections of the external canal are of daily occurrence; in five years my associates and I have had four of the mastoid and two of the middle ear.

The pathologic features are as follows: There are multiple perforations, seldom marginal, through most of which polyps protrude, especially during healing. The drum membrane is gray, thickened and less sensitive than normal. When the drum is opened, the mucosa is found swollen, with obliterations of the cavity. On removal of the mastoid cortex, the cavity is characteristically filled with a pigmented fungous growth, contrasting with the bone. Coalescence is not consistent, and in old cases the membrane will be polypoid. In our 4 cases the process was limited to the pneumatic spaces. The radical cavities from mastoidectomies frequently become secondarily contaminated in the southern climate. This paper lists eight types of yeastlike fungi, three types of molds and two high bacterial forms. Diagnosis by smear is hazardous; culture is slow; cutaneous tests are practical but uncertain. During acute stages bacteria predominate; during chronic stages, fungi.

As to treatment, potassium iodide, thymol and sodium benzoate are best for systemic medication and are useful locally in salves or solutions; a preparation of bismuth and crystal violet (bismuth violet), metacresol acetate (cresatin), silver picrate and solutions of copper sulfate also. A melted salve containing benzoic acid 12 per cent, salicylic acid 6 per cent and monochlorthymol 0.25 per cent, liquefied and instilled, with prevention of bubbles and pockets, seems the best; it is used every second or third day at first; later, at longer intervals. Propylene glycol may prove a better vehicle. Chemotherapy diminishes the bacterial element and the discharge.

Six cases are reported in detail.

Allergic patients become sensitive instead of immune, and their infections tend toward chronicity. They must be desensitized to the fungi.

When the mastoid process is involved, radical mastoidectomy is necessary.

DISCUSSION

DR. CLARENCE H. SMITH, New York: The fungi commonly found in the ear are Monilia, Aspergillus, Penicillium and Achorion. They may be cultured from the surface of normal skin and may be merely secondary invaders. They occur more commonly in a warm, moist climate, and therefore Dr. Shea undoubtedly has a wealth of material to draw from.

For years I have used, with uniform success, a 2 per cent solution of salicylic acid in alcohol yet, according to Allen and Wright, this solution ranks thirty-ninth among forty-three substances employed. Perhaps the exfoliation produced by the salicylic acid and the drying and solvent effect of the alcohol are more valuable than a fungicidal-germicidal action. Does it really matter how the cure is produced if the patient is relieved simply and promptly?

Many others have used alcohol alone with success. According to William D. Gill, metacresyl acetate (or cresatin) heads the list in degree of effectiveness. He advised the addition of a vegetable oil to prevent undue inflammation of the

surface epithelium. He recited how he used this in the manner described on a patient in whom severe dermatitis of the pinna and surrounding skin had developed.

When the middle ear and mastoid process are affected, treatment is much more difficult than when the external canal is involved, and it seems that radical surgical intervention is inevitable sometimes. I wonder if Dr. Shea ever tried roentgen radiation for its restraining effect on sporulation.

Dr. Charles D. Blassingame, Memphis, Tenn. (with slides): The salve is heated in this porcelain dish (slide) until it is quite thin; it will not burn if injected directly in. If the syringe is put into the sterilizer, it takes a day or two to get the debris out.

I now use a Luer syringe with a rubber extension (slide) to get the material in. The medicine dropper will not do it.

With the syringe in this position (slide) and the tube down to the tympanic membrane, the canal fills from the bottom.

As the tube is withdrawn (slide), the canal is filled. I have quit worrying about how to treat fungous infections.

Dr. H. Marshall Taylor, Jacksonville, Fla.: Different species of Aspergillus are common in Florida. In one day I have seen in my office patients infected with Aspergillus glaucus, Aspergillus niger and Aspergillus flavus. I can make the diagnosis now without a culture. In the North these species are somewhat rare. One culture in a tube was misplaced in the safe for four years. It was then cultured and found still active. I have never seen a fungus infection that was not cleared up promptly with 1 per cent salicylic acid in 65 per cent alcohol. The infected areas should be treated once or twice a day for the first two or three days. The patient then continues using the drops once daily for a month. It is necessary to see the patient after the acute stage has subsided to remove the parchment-like substance which will otherwise fill the canal. Dr. Shea spoke only of mycosis; I think the majority of the infections are due to mixed organisms. I have never seen any cases of multiple perforations. In the cases of infection with A. glaucus this organism may be mixed with Bacillus pyocyaneus, but I think virtually all are mixed infections. I have never found it necessary to use iodides over a period. In the mastoid process everything seems propitious for mycotic growth, but I have never recognized a mycotic infection of the mastoid antrum.

Actinomycosis of the Temporal Bone. Dr. John MacKenzie Brown, Los Angeles.

Actinomycosis is an infectious granuloma caused by any one of several species of fungi of the genus Nocardia or Streptothrix.

In 1877 Bolinger described lumpy jaw in cattle. In 1882 Ponfick established the unity of bovine and human infections by this organism. In 1885 John B. Murphy described the first case seen in America.

In 1910 Urbantschitsch reviewed the literature of actinomycosis of the middle ear (6 cases). He stated that extension was usually directly by way of the mouth and the eustachian tube or hematogenous.

Dr. Joseph C. Beck encountered a case affecting the petrous pyramid in a routine autopsy in the Chiari clinic in Prague, Czechoslovakia.

In 1937 there were two reports from Germany.

The disease is acquired when infected vegetable matter gains access to the mucosa or the cutaneous tissues. It is not frequent among those in contact with animals, such as employees in packing houses, but one commonly finds a history of contact with straw or other vegetable matter.

In man the disease may affect all parts of the body but is more common about the head and neck. Abdominal lesions are next in frequency, and pulmonary and bone lesions less common. In cattle the infection usually starts in the buccal mucosa and extends to the mandible, causing lumpy jaw; the organism is forced into the dental tissues by hard particles of food during chewing and grows through the tissues between the teeth and the alveolar process, progressing to

the bone marrow. Later internal or external fistulas are formed. In man the infection tends to avoid the bony structures and spreads through the soft tissues.

From a histologic standpoint the early lesion roughly resembles a tubercle but breaks down quickly into fairly large abscess cavities. The diagnosis depends on finding the characteristic sulfur granules in the discharge.

The microscopic picture consists of three zones: (1) a central zone of compact pigment; (2) an intermediate amorphous and deeply pigmented area; (3) an outer fringe of striated groups of mycelial hyphae.

The organism is often difficult to isolate. In all cases reported in this paper the diagnosis was made post mortem.

There is a high mortality. Iodides and specific vaccines have been of some benefit. Thymol has been useful, but no agent has been found adequate in a large number of cases. There are reports of success with sulfanilamide. At the Los Angeles County Hospital 3 patients have been cured since 1940 with this drug, orally administered. Two cases are reported in detail.

In both cases the course was relatively afebrile; in both, the retropharyngeal and parapharyngeal spaces were involved in continuity with the temporal bone, and both patients died with intracranial symptoms. Despite diligent search, no antemortem diagnosis was made.

Summary.—In about 60 per cent of patients with actinomycosis the disease affects the head and neck. In man the spread is by fistulas and abscesses in the soft tissues, and in bone it is by osteomyelitis. Any progressive chronic infection of the head or the neck with cultures negative should arouse suspicion, and a search should be made for fungi.

The excellent results obtained in 3 cases of cutaneous actinomycosis offer a hope of cure if the disease is recognized early.

DISCUSSION

Dr. T. E. Carmody, Denver: I have not seen the infection of the temporal bone, but the closeness of association with the jaws has in it a resemblance to a middle ear infection. My associates and I find the sulfur granules frequently before some form of treatment has been instituted. The ordinary pathologist will not find them frequently. They have been found for me by Drs. Stiles and Davis of our bureau of animal industry. Dr. Brown says they are not found frequently in farmers or in workers in packing houses. In my case 1 the patient, a nurse, probably got her infection from vegetables in the garden, and in case 2 the patient, a grocery clerk, admitted that he chewed small portions of wheat. The infection crowds in between the teeth; in both my cases the trouble began in the oral cavity. According to Dr. Davis, there are two infections: that due to an actinomycete, a fungus, and that due to an actinobacillus. Another confusing infection is coccidiosis, which occurs especially in the San Joaquin Valley, in California.

A friend of mine suggested the use of compound solution of iodine (Lugol's solution), because it does not cause swelling of the face. The patient in case 1 received about 80 ounces (2,365 cc.) of compound solution of iodine before permanent cure was obtained. The patient in case 2 was first treated with thymol crystals after operation, with no result, then a crystal of copper sulfate was packed into the wound, with temporary improvement; sulfonamide compounds gave no result. Thereafter he used compound solution of iodine until three months ago. He reported himself entirely well except for ankylosis of the jaw. The actinomycete affects the bone; the actinobacillus, the soft tissues; probably in many cases one deals principally with actinobacillosis. (Slides were shown illustrating the fungus, the bacillus, the characteristic granules, patient 1 and patient 2; also, one showing the ankylosis, believed to be fibrous, as it is improving, and one showing the mouth closed.)

Dr. William H. Johnston, Santa Barbara, Calif.: Actinomycosis of the temporal bone is rare; cases were reported by fourteen authors between 1890 and

1939. External infection of the auricular region is not uncommon. It is important that it may extend by contiguity. Such a case came under my observation many years ago. Organisms were found in the pus from fistulous tracts, and the response to iodine therapy was satisfactory. The process is characterized by suppuration, granulation tissue and necrosis. The pus contains the sulfur granules. Spread is by contiguity, and intracranial complications may develop without much warning. Recent investigations show that Actinomyces bovis is a normal saprophyte of the human alimentary canal and can be demonstrated in the mouth and on the tonsils. It is easily understood how such infections may develop after extraction of teeth, as in a case of Dr. Brown's. The diagnosis cannot be made by clinical symptoms alone; the organism is found in the pus but is not always found. A gelatin culture medium seems as satisfactory as any. The prognosis seems to depend on the virulence of the organism. Some observers still believe that iodine is a specific, administered locally and internally. Radium has been used successfully for the granulations. One patient recovered promptly on the administration of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole). Perhaps chemotherapy will replace the use of iodine. The proximity of the intracranial structures to the middle ear and mastoid process makes involvement of this region a grave condition.

Dr. H. M. Goodyear, Cincinnati: I wish to mention a case of actinomycosis of the parotid gland. The bony tissues were not involved, the antrum was clear and there was no disturbance of the jaw. There were multiple fistulas in the skin which would clear and break down repeatedly. There was also one in the mouth, and the diagnosis was not made for some months, although the material was cultured. It cleared rapidly under chemotherapy and use of roentgen rays.

Dr. John M. Brown, Los Angeles: I am glad to learn of the treatment with compound solution of iodine. In the first case in which I tried chemotherapy the infection cleared up in about one mouth. I still believe that type of therapy is the best. If involvement of bone has occurred and the bone cannot be eradicated, the physician is in trouble. A veterinarian told me that the iodides were valuable in the treatment of cattle but were of little value if fistulas were present.

Otitic Meningitis: A Review of the Literature and a Report of Results Five Years Before and Since the Introduction of Chemotherapy.

Dr. Henry L. Williams, Dr. Wallace E. Herrell, Dr. Alex E. Brown, Dr. James W. Kernohan and Dr. Henry P. Wagener, Rochester, Minn.

Until 1937 streptococcic meningitis was considered a highly fatal disease. In the previous thirty-four year period only 66 cases in which recovery occurred were reported. Caussé and Gisselbrecht early in 1936 reported the first case of hemolytic streptococcic meningitis in which chemotherapy (prontosil [the hydrochloride of 4-sulfamido-2',4'-diaminoazobenzene]) was used. In 1937 these drugs came into prominence, and since then many cures have been reported.

Two types of otitic meningitis occur clinically: (1) the fulminating type, in which the meninges are invaded simultaneously with invasion of the ear and in which meningitis is probably secondary to bacteremia; (2) a type which develops by transmission from contiguous disease. The distinction between cases in which bacteria can be cultured from the cerebrospinal fluid and those in which the fluid is bacterially negative is of great clinical importance because of the difference in the mortality rates which accompanied those two types before the introduction of chemotherapy. Courville and Nielsen preferred the classification of Goerke:

A. Direct or primary meningitis (direct extension from the temporal bone)
   1. Hematogenous form, extension by way of the pial veins
   2. Tympanogenous form, extension by preformed paths or pathologic paths
   3. Labyrinthogenous forms

B. Indirect or secondary meningitis (extension by way of other intradural complications)

1. Direct transmission from thrombosis of the lateral sinus; subdural infection; abscess of the brain

2. Symptomatic meningitis associated with the aforementioned conditions

Cawthorne qualifies meningitis as serous, cellular or bacterial, based on examination of the cerebrospinal fluid. A lengthy description of the route of advance to the meninges is summed up as follows: Acute otitis media may cause meningitis within hours by spread through certain preformed spaces, within days by spread through veins, and within weeks by destruction of intervening bone.

Meningitis is a disease of the arachnoid and cerebral tissues. In these tissues nonreversible changes take place rapidly. The reaction in the arachnoid meshes may localize the lesion. Eagleton found the venous pathways to be chiefly the veins of the internal auditory meatus, the venules within the saccus endolymphaticus and those of the jugular bulb. The venous walls may break down, producing local abscess, frequently adjacent to the pyramid. A mistake, he said, would have been avoided if he had appreciated the significance of a positive blood culture without thrombosis of the venous sinus. The literature covering the histopathology is further elaborately quoted.

Diagnosis.—The typical onset is characterized by severe headache, mostly frontal and occipital but undergoing extension. This is followed by dulling of the sensorium and delirium, at first in sleep, or somnolence alternating with delirium. The headache persists even in stupor, and the patient makes painful grimaces when his head is moved. When fully conscious, he complains of vertigo and sensitiveness to light and noise. Vomiting is often present. According to Eagleton, vomiting is clinical evidence of the date when infection enters the meninges, and absence of headache and presence of drowsiness with intermittent rotary nystagmus indicate that the disease is spreading by septic periphlebitis. Further signs are rigidity of the neck and the Kernig and Brudzinski signs. Increased pressure of cerebrospinal fluid with a normal cell content was considered the expression of a protective reaction. A count of 100 cells per cubic millimeter in a case of aural disease suggests beginning localized meningitis. Organisms are evidence of generalized septic meningitis. Kopetsky and Fishberg have attached significance to chemical examination of the spinal fluid.

In the distinction of tuberculous otitic meningitis we believe a relative increase in lymphocytes, as opposed to an increase in polymorphonuclear leukocytes, is of diagnostic significance. If pellicle formation is present, Mycobacterium tuberculosis may be demonstrated therein.

The pupils are usually contracted at first; later, dilated. Ptosis and paralysis of the ocular muscles suggest involvement near the apex of the petrous bone. Eagleton mentioned transient rotary nystagmus as indicative of meningitis localized in the pontile cisterns. Transient anesthesia of the homolateral cornea may indicate invasion of the middle fossa. Hemianopic indentations of the visual fields may occur from edema involving the homolateral optic tract. Definite hemianopic defects more likely indicate abscess of the temporal lobe. Choked disk may occur, but less often than in other intracranial conditions.

Treatment requires three major procedures: (1) drainage of the meninges, (2) eradication of the primary focus and (3) chemotherapy.

We prefer repeated lumbar puncture and drainage. In cases of fulminating meningitis, drainage may be avoided if chemotherapy is started early. If there are signs of an abscess of the brain, drainage of ventricles may be preferred.

As to surgical eradication, opinions differ as to the procedure. Neumann advised exenteration of the mastoid cells plus exposure of the dura of the middle and the posterior fossa. Others added to this, continuous drainage of spinal fluid. For nonfulminating conditions Lewy and Hagens advised exenteration of the mastoid cells and uncovering of the dura of the middle and the posterior fossa

without waiting for the report as to the culture. They followed the original recommendations of Neumann in addition to recognizing the importance of foci in the petrous pyramid. Long pointed out that, in spite of chemotherapy, adequate surgical drainage is important to prevent recurrence. Osseous lesions are not clearly influenced by the sulfonamide compounds. Kopetsky opposed surgical drainage of the meninges and expressed the belief that better results are obtained by transfusion of small amounts of blood. In the opinion of Friesner and Rosenwasser, surgery offers little for the fulminating type. They found suppuration in the petrous pyramid the most common pathway to the meninges; suppurative labyrinthitis has no tendency to localize. They expressed a preference for lumbar puncture rather than incision and drainage of the meninges. Sulfonamide compounds are useful only if the primary focus has been removed.

Brunner studied 70 cases in which recovery took place after the use of sulfonamide compounds and compared them with 64 cases in which recovery occurred without chemotherapy. He concluded that chemotherapy does not replace surgical treatment. He quoted Jansen's axiom "The first symptom of meningitis is the last summons to operate."

In our opinion, complete mastoidectomy should be done for suppurative disease of the temporal bone whenever evidence of dural irritation or of irritation of the meninges is observed. The use of sulfonamide compounds should be instituted prior to operation. In addition to drainage of the involved pyramid, the dura of the temporosphenoidal bone should be uncovered down to the labyrinthine capsule. The uncovering should go as far as is necessary to reach normal dura. For suppurative labyrinthitis an operation of the Neumann type should be done. We do not advocate incision of the dura.

Chemotherapy to be successful must be adequate. Hewell and Mitchell reported 8 cases of meningitis with positive blood cultures. In only 1 case did recovery take place, and in this case the concentration of the drug in the blood was 20 to 25 mg. per hundred cubic centimeters. In the fatal cases it was less than 15 mg. The toxic effects of the drug are ignored, except those which in themselves might terminate life, such as agranulocytosis and complete suppression of renal function. Sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) is preferred for all pneumococcic infections and for all unknown infections. Sulfanilamide is preferred if the organism is identified as a hemolytic streptococcus. Adults receive 1 grain (0.064 Gm.) per pound of body weight up to 120 grains (7.76 Gm.), one half or three fourths of which is given at the first dose. Thereafter adjustments are made according to the concentration of the drug in the blood. If the patient is comatose or vomiting, sodium salt of sulfapyridine is used, 100 cc. of a 5 per cent solution being administered intravenously, or sulfanilamide is used, 500 cc. of an 0.8 per cent solution being injected. We feel that 16 to 20 mg. per hundred cubic centimeters is the necessary concentration of the drug. We continue the daily administration of the drug, but reduce the doses to approximately 60 grains (3.88 Gm.), for three weeks after apparent cure. Otherwise recurrence is likely.

The action of sulfapyridine in the blood is merely bacteriostatic. Leukocytes are necessary to kill the bacteria. This destruction is promoted by antipneumococcic serum. If the response to chemotherapy is not satisfactory, the patient should be given the benefit of antiserum in addition. Fluid should be administered to maintain a urinary output of 1,500 cc. per day. Frequent transfusion of small amounts of blood is of value. Chemotherapy is most effective in a medium rich in oxygen. Oxygen therapy is considered as a useful adjunct to chemotherapy. Maintenance of nutrition is important, and we administer with each liter of dextrose vitamins B and C. Close cooperation between the internist and the otolaryngologic surgeon is paramount.

We have studied 46 cases of otitic meningitis observed in five years before chemotherapy (group 1) and 24 cases observed in five years since then (group 2). In some cases smear and culture failed to disclose micro-organisms. In thirty cases suppuration of the petrous apex was associated with meningitis. In 2 cases petrositis remained dormant for eighteen months and in another for ten years.

In group 1 were 7 cases in which fatal otitic meningitis occurred subsequent to mastoidectomy. Previous suppurative labyrinthitis increased the operative risk. Meningitis occurred before mastoidectomy in 9 cases, with 2 recoveries. In 20 cases there were clinical evidences of meningitis but organisms could not be demonstrated. Fourteen patients recovered.

In group 2, in spite of a greater number of hospitalized patients treated for middle ear and sinus disease, meningitis developed in only 24 patients; in 6 it was of the fulminating type; of these, 2 recovered. In all, 11 recovered and 13 died. In the cases of 4 of the 13 chemotherapy was used too late.

We feel that in most cases in which the patient died, adequate chemotherapy was not used. However, 1 patient with a green-producing streptococcus died in spite of a high concentration of sulfapyridine. It is interesting to note that of 9 patients with positive cultures who did not have the benefit of chemotherapy, only 2 recovered. Of 14 with positive cultures who received such therapy, 8 recovered. Operation is particularly necessary in cases of infection with Diplococcus pneumoniae, because of the formation of secondary foci in the meninges. Four cases in which meningitis developed a month after otitis media had apparently been cured with a sulfonamide compound illustrate the masking effect of such therapy. It is difficult to discover whether the numerical reduction in the incidence of meningitis is due to diminution in the virulence of the infection or to its prevention by chemotherapy. We favor the latter hypothesis inasmuch as there has also been a diminution in the incidence of otitis media.

DISCUSSION

DR. DANIEL S. CUNNING, New York: This is a thorough, complete and painstaking review of the published material on this subject.

I am glad to note that the authors resort to spinal taps as often as necessary, and I believe that this procedure alone does a great deal toward reducing the cerebrospinal pressure and that, besides helping the patient against the infection, it makes him more comfortable. When the infection is severe, my colleagues and I tap twice a day, and if the pressure is marked, three or four times a day.

I wish to emphasize the necessity of cleansing the mastoid cavity thoroughly. There are two points as to which I cannot thoroughly agree: First, the authors are opposed to slitting the dura. On several occasions I believed this was the deciding factor in the patient's recovery. In 1939 I described the treatment of 14 consecutive cases with 11 recoveries. In 4, in spite of thorough mastoidectomy and intensive treatment with sulfonamide compounds, the outlook seemed hopeless. Then the dura was slit by several longitudinal incisions, allowing free flow of spinal fluid, and within a short time the patient's condition improved, with full recovery following. Dr. Marvin Jones recently had a patient recover from meningitis due to Pneumococcus type III, in whose case he felt the slitting of the dura played no small part. I do not believe it makes much difference where the dura is slit, provided one obtains a free flow. In cases in which an operation has been done, sulfonamide compounds given properly and the patient shows no improvement, why not slit the dura? One has nothing to lose and possibly a great deal to gain. I have never seen a bad effect from this procedure.

The second part of the presentation with which I do not agree is that on the use of sulfapyridine. We have been using sulfadiazine (2-[paraaminobenzene-sulfonamido]-pyrimidine) and believe it is the drug of choice. It acts well in combating practically all organisms. A higher concentration can be carried in the blood and spinal fluids than of any other sulfonamide compound. The majority of workers agree that sulfadiazine is less toxic than any of the others in this group.

DR. WILLIAM E. GROVE, Milwaukee: When this paper is published, the readers will see that it is exhaustive and well arranged. The most interesting fact developed by the survey of the pathology of meningitis in cases of otitis is that the cortex of the brain becomes inflamed and infected very early in the course of purulent meningitis.

Vomiting in the course of otitic suppuration has long been considered suggestive of meningeal irritation, but the suggestion that headache of any sort in association with otitic infection should put one on guard is of extreme importance. These two symptoms may precede by hours or even days the more classic signs of meningitis and should suggest the pushing of adequate therapy at this time. At least they should suggest early examination for changes in the spinal fluid.

Eagleton's statement, quoted by the authors, that a count of 100 cells per cubic millimeter in the spinal fluid of a patient with otitic infection suggests beginning localized meningitis sounds generous to me. I prefer Lund's, to whom a count of 10 or more cells spells trouble. The position of the Mayo group corresponds closely to my feeling.

The ophthalmologic observations indicating changes in the fundus in 20 per cent and other ocular abnormalities in 45 per cent are interesting, but these symptoms usually appear late or are evidence of other intracranial disease.

The authors speak of three general principles: (1) drainage of the meninges; (2) eradication of the primary focus; (3) use of chemotherapy. I should like to suggest a reversal of the order: (1) eradication of the focus and at the same time chemotherapy; (2) spinal drainage if any drainage at all. It has never seemed feasible to me to drain the arachnoid space through repeated lumbar punctures or by continuous spinal drainage.

I like the statement of the authors that with fulminating meningitis spinal drainage can be avoided if chemotherapy is started early. Chemotherapy should be started as soon as there is any suspicion of a meningitic process. This, together with full eradication of the suppurative lesion, should obviate the necessity for such a doubtful procedure as spinal drainage. This has nothing to do with slitting the dura.

I see no reason for omitting the eradication of the primary focus because one has a new weapon in chemotherapy. Chemotherapy has no efficacy in handling a bone-locked collection of pus. Therefore, I shall continue to do a full surgical clean-up as soon as meningitis is suspected, if it has not already been done.

It is fortunate that hemolytic streptococci and type III pneumococci are the predominating organisms among those found in otitic meningitis, for otologists have specific bacteriostatic agents for these two invaders. I also agree that it is necessary to keep up the administration of small doses of the drug for weeks after apparent cure of the meningitis. I have seen recurrence when the administration of the drug was discontinued too soon. Thirty-five per cent of recoveries in the group of cases preceding the employment of sulfonamide compounds is too good to be true if those 46 cases were all instances of true purulent meningitis. I suspect that many of them were cases of so-called protective meningitis.

The analysis of the cases observed during the period of chemotherapy is excellent and shows a gratifying decrease in mortality.

DR. HENRY M. GOODYEAR, Cincinnati: A symptom one can often rely on, and which sometimes is the only symptom, in making an early diagnosis of meningitis in a baby, is sustained ankle clonus.

I believe most otologists agree that when suppuration of the temporal bone is present with meningitis an operation is indicated. Free exposure of the dura in the middle and the posterior fossa, if indicated, should also be done. Such decompression may have a valuable effect in delaying rapid decomposition of the brain tissue and may also help to relieve pressure on the veins in this area.

There is no continuity of lymphatics between the extracranial and the intracranial structures, yet in a recent textbook it was stated that infection may extend from the ear intracranially through the lymphatics.

It is in the treatment of the acute fulminating type of meningitis occurring in the first few days after the onset of an acute infection of the middle ear that there is disagreement as to the time of operation. To incise an acutely infected finger or furuncle of the nose before nature has had some opportunity to produce a walling off and localization of the infection would be considered hasty and bad

surgical practice. To invade surgically the mastoid cells in an acute hyperemic stage is equally bad. It tears down nature's first wall of defense and may spread the infection. The shock of the anesthetic and the operation may turn an early meningismus or a localized meningitis into a diffuse and suppurative affair. If a mallet and a gouge are used, the concussion of the labyrinth, with petechial hemorrhage, can well be pictured.

I do not feel that one gains anything by operating on an acutely infected middle ear if meningitis has shown up in the course of two to four days. If the mastoid is opened in such a case, one usually finds hyperemia and perhaps some mucopus in the antrum but no necrosis of bone. One has not even approached the most likely and frequent area of invasion—the epitympanic space which is traversed by the petrosquamosal suture, which is present until the age of 2 years and occasionally persists in adults. This suture presents a direct communication between the dura and the mucous membrane of the middle ear and frequently serves as a passageway for a large vein connecting the lateral sinus with the superior petrosal sinus. In most instances acute fulminating meningitis is streptococcic in origin and as such has a predilection for veins; one cannot hope to ward off a serious complication by premature surgical intervention.

A patient with meningitis developing between the seventh and the tenth day after the onset of acute otitis should be operated on. I should like to emphasize the importance of ventricular puncture, which in extreme cases may be a life-saving procedure.

Sulfanilamide is the most satisfactory drug when the infection is due to hemolytic streptococci, but when the bacterium has not been definitely determined, sulfadiazine is preferred and the sodium salt is given intravenously. This drug cannot be given subcutaneously. If the infection is due to pneumococci, sulfadiazine is the first choice, but will be less satisfactory if the pneumococci are of type III, which is a poor producer of antibodies. This is also true to a less degree for pneumococci of type VIII, with which type-specific serum is most helpful. Frequent transfusion of small quantities of blood may be necessary, and oxygen is helpful.

Sulfanilamide gives a concentration of 1:1 in the blood and the spinal fluid; sulfathiazole, 3 in the blood to 1 in the spinal fluid, and sulfadiazine 3 in the blood to 2 in the spinal fluid. Sulfadiazine is the drug of choice, except in the beta type, and with it concentrations up to 20 mg. in the blood may be maintained for long periods without apparent damage.

Sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) is apparently more damaging to the kidneys, especially in adults. The chief objection to sulfadiazine is the cost—$60 a pound. Bacteria will live in test tubes with the sulfonamide compounds and with iodized oils, yet strangely enough the usefulness of these chemicals in their respective fields is assured.

Dr. Alfred Lewy, Chicago: In the consideration of the cases in group 1, before the use of chemotherapy, did Dr. Williams separate those in which the culture of the spinal fluid was positive from those in which it was not? Dr. Goodyear's suggestion that ankle clonus is a diagnostic feature when there are no others reminds me of a case in which a child with a high temperature and no neurologic signs or symptoms except vertical nystagmus was found post mortem to have meningitis due to a hemolytic streptococcus infection.

Dr. Harry W. Lyman, St. Louis: In regard to the recoveries in 33 per cent of cases of meningitis before the sulfonamide compounds were used, the proportion may depend on the difference between the view of the pathologist and that of the clinician. In regard to clinical meningitis my colleagues and I feel that if organisms eventually show up in the spinal fluid our patient will die. So before the sulfonamide compounds came into use we operated in those cases as promptly as possible, even on the preliminary symptoms. Nowadays I think most otologic surgeons are agreed that it is safer for the patient if the cleaning out of the original focus is postponed until the patient is thoroughly saturated with the drug.

The object of repeated spinal punctures is to keep down the spinal fluid pressure and avoid damage to the brain tissue. I believe in giving large doses of sulfonamide compounds, with the dosage controlled by the concentration in the blood and the spinal fluid. It should be kept at the highest level commensurate with safety. The chemotherapy should be kept up for at least three weeks after the patient is clinically well.

In using sulfonamide compounds in the treatment of erysipelas, the erysipelas may clear up beautifully and the patient be sent home only to come back in ten days or two weeks with large cold abscesses in various places. The fluid from these abscesses contains viable hemolytic streptococci, showing that the drug has kept the growth of these organisms in abeyance but that nature had not yet had a chance to destroy all of them. They are able to grow again when the drug is withdrawn.

Our experience has been more favorable with sulfadiazine than with the other sulfonamide compounds.

Dr. Wallace E. Herrell, Rochester, Minn.: I do not want to leave the impression that sulfadiazine is not as good as sulfapyridine. It just happens that during the period in which this review was carried out the drug used was sulfapyridine sodium. Nevertheless, we have seen every toxic manifestation of chemotherapy with sulfadiazine but I believe it will replace sulfapyridine.

Regarding the separation of the cases in which the culture of the spinal fluid was positive from those in which it was negative, I think that will probably be found to have been done in the paper, which was not read in its entirety.

Dr. Lyman's suggestion of saturating the patient before surgical intervention I think is important, but in the case of pneumococcic meningitis it is probably not wise to temporize, as foci will be set up earlier than in the case of hemolytic streptococcic meningitis.

Ossiculectomy in the Treatment of Chronic Suppurative Otitis Media.
Dr. Frederick T. Hill, Waterville, Maine.

In 1873 Schwartz proposed removing the drum membrane and the malleus to improve hearing. Later he extended the use of this procedure to chronic suppuration. In 1890, Ludwig removed the incus, and Stacke added removal of the attic wall.

The objective of surgical procedure is free and permanent drainage. The nature and the extent of the pathologic process should determine the surgical procedure. In the absence of impending complications it is best to err on the side of conservatism. More radical measures can be applied later. The complicated anatomy of the epitympanic space is a barrier to free drainage.

The proper cases for ossiculectomy are limited in number. Operation is futile if the roentgenogram shows an enlarged antrum or a cholesteatoma, or if there are signs of impending complications.

The technic is described. With proper selection of cases, a fair proportion of satisfactory results is obtained. More radical surgical procedures can be done for the failures. A series of cases is reported.

DISCUSSION

Dr. Harold Tobey, Boston: Ossiculectomy usually necessitates complete removal of the drum membrane. The head of the malleus may be removed, but there are usually necrosis of the incus as well as of the malleus and considerable destruction of the drum membrane. The removal of granulations in the attic through a superior perforation is not included in this discussion.

The indications are well presented, but I wish to emphasize two points: 1. The preservation of any residual hearing is relatively unimportant, as is true of the radical mastoid operation. It is yet to be proved that in any case operation by any method will definitely increase or decrease the hearing. The goal

to be aimed for is the safety and well-being of the patient. 2. The roentgenograms and their interpretation are perhaps the most important guides to the choice of operation. Both lateral and anteroposterior views are necessary. The lateral helps to visualize any cells in the zygoma, the retrofacial area or other regions of the mastoid process. The anteroposterior positions give a truer estimate of the size of the antrum and reveal cholesteatoma, if this is present.

The contraindications are: (1) persistent and recurring headaches, denoting probable exposure of the dura; (2) recurrent attacks of vertigo even in the absence of a positive fistula test; (3) a positive fistula test even in the absence of vertigo; (4) residual cells in the mastoid process; (5) an enlarged mastoid antrum filled with cholesteatoma.

I prefer general anesthesia and I prefer the Blake mastoid curet in several sizes. If necessary, the removal of the posterior wall can be continued outward and downward external to the facial nerve and fairly complete radical mastoidectomy performed through the canal. In the same manner the annulus can be removed and the hypotympanum curetted. By careful placing of this curet, the side of which lies on the floor of the aditus, and by using an upward and outward movement one can make sure that the instrument is always moving away from the facial nerve, the posterior portion of the bowl causing no trauma. Healing is much more rapid and complete than in the radical operation. There were 3 cases in which temporary facial paralysis developed on the fourth or the fifth day, which followed insertion of an alcohol wick into the middle ear.

In only 1 of my 25 cases was a secondary radical operation required. This necessity arose three years later. When the second operation is performed, the patient should be under the close observation of the operator and should not be lost sight of in the clinic. The endaural approach is extremely satisfactory in the cases in which the drum is nearly intact and when the infection is confined to the attic and the antrum. The posterior wall of the external canal can be left intact, the incus can be exposed and the head of the malleus removed. Following this, the posterior membranous wall of the external canal can be freed by an incision extending from the upper posterior portion of the drum membrane outward to the original endaural incision. This frees the posterior membranous wall which can be utilized as a flap.

Dr. D. Harold Walker, Boston: Dr. Hill brings up some forgotten things which may well be cited now and then. One not mentioned was the question of reinfection of the eustachian tube from the nasopharynx. It is important to know that there may be a tubal infection and that the discharge is not necessarily coming from the antrum and mastoid process.

Another cause of a chronic discharge to be considered is mycotic infection and not necessarily chronic osteitis in the ossicles and surrounding parts.

Dr. George M. Coates, Philadelphia: It seems to me that this is not an obsolete operation and that it needs reemphasis. I still practice it occasionally and still teach it. The cases must be carefully selected. This operation should be employed only when the disease is limited to the middle ear, including the attic and perhaps the antrum but not the remaining mastoid cells. Roentgenologic diagnosis is difficult in many of these cases because of the sclerotic cortex. All chronically discharging ears should be carefully studied before they are subjected to operation, and ordinary methods tried for control of the disease.

Simple ossiculectomy is not difficult. The operation suggested by Dr. Tobey is more than simple ossiculectomy. Simple ossiculectomy with removal of some of the epitympanic wall is often sufficient. I always try to close the eustachian tube to prevent reinfection. This, however, cannot always be done.

If there is necrosis of the ossicles, they have no further function in regard to hearing. The procedure should not be done in the face of acute exacerbation, oncoming intracranial complications or any other of the contraindications which were mentioned.

If the disease is limited to the mastoid process and there is relatively good hearing, it seems to me that a modified radical operation should be the choice.

If there is considerable involvement of the mastoid process plus destruction of the middle ear, a radical mastoid operation is necessary.

If the case has not been properly selected and the ossiculectomy fails, at least one has done part of the radical operation and it can be finished later.

I prefer to use general anesthesia. One is not sure that the patient will be quiet under local anesthesia.

Meticulous after-care is perhaps the most important part of the therapy.

Dr. James A. Babbitt, Philadelphia: Successful management of an infection of the middle ear depends on judicious use of whatever method a skilful technician finds is best adapted. I prefer to do it by draining through two outer quadrants, and in the majority of my cases the balance has been in favor of a little better hearing. Free drainage of that outer area is perfectly safe. In few cases was there any labyrinthine trouble.

I have a divided view in regard to closing the eustachian tube. With complete ossiculectomy there should be no fear of reinfection of the eustachian tube if the postnasal space is properly cared for. I had a curious experience last year, operating on my dear old chief, Dr. Randall. It was a radical mastoidectomy and the hearing was brought up from a 35 or 40 decibel loss to nearly normal by getting the eustachian tube open after it was once closed. I have seen that happen time and time again.

Dr. Frederick T. Hill, Waterville, Maine: In reply to Dr. Tobey as impersonated by Dr. Walker, I prefer local anesthesia. I think that with careful preoperative medication, local anesthesia induced according to Neumann's technic and, in addition to this, Bonain solution (phenol, cocaine and menthol in equal parts) packed into the middle ear, the anesthesia is quite satisfactory.

I use the Citelli forceps to take down the outer attic wall.

I am grateful to Dr. Walker and Dr. Coates for the warning about the importance of nasopharyngeal foci.

Careful selection of patients is required, and careful observation and treatment should be maintained for at least a year.

I formerly curetted the eustachian tube. I now feel that if I take care of the nasopharynx, it makes little difference whether the tube closes or not. I agree with Dr. Babbitt about the after-care. One must adapt the procedure to the individual condition, striving for adequate drainage.

Residual Hearing. Dr. C. Stewart Nash, Rochester, N. Y.

This study deals with acoustically handicapped children who possess some degree of residual hearing. The child who hears the conversational voice at 20 feet (6 meters) may be managed in a favorable seat in a regular school. If the loss is greater, the child must be put in the special class or in full attendance at a school for the deaf, according to the severity of the loss.

With a hearing loss up to 30 decibels a child will have natural speech; with a loss of 30 to 50 decibels, natural to partially defective speech; with 50 to 90 decibels, fair to poor speech, and with 90 or more decibels, poor speech. The Rochester School for the Deaf divided 218 pupils into four groups according to the number of decibels of hearing loss: group 1, up to 40; group 2, 40 to 60; group 3, 60 to 80, and group 4, 80 to 120. The last group embraced 65.6 per cent of all the pupils.

The children in group 1 rarely require instruction in the school for the deaf. As to those in group 2, much depends on the character of the loss. If the loss is greatest in the high frequencies, there is difficulty in interpreting spoken language. If the impairment is congenital, the speech will be defective. The pupils in group 3 are unable to interpret spoken language without training unless they have acquired an understanding of language before the onset of their deafness. In group 4 many have benefited by auricular hearing. The principal purpose in this group is to stimulate a sense of hearing sufficient to recognize to some

extent speech rhythm, inflection, phrasing and emphasis and to acquire some discrimination of vowels. Deafness for high frequencies implies usually imperfect hearing of consonant sounds, especially of final consonant sounds. With a loss of between 30 and 50 per cent the mentality of the child is a determining factor in the acquisition of speech.

I have inquired of seven schools, "Does a small amount of residual hearing give any advantage over the child with no hearing at all?" "How much residual hearing is required?" Here are some of the answers:

1. "Unless there is 45 per cent of residual hearing, it is of little benefit."

2. "A bright child with an 80 per cent loss should be in a hearing aid room."

3. "Any amount of residual hearing should receive training."

4. "It is impossible to have any fixed criterion—results are influenced by intelligence, type of hearing loss, range of hearing, age of onset of deafness, environment, previous educational opportunities."

5. "Any deaf child who has 25 per cent of hearing left can be helped by a good hearing aid."

6. "If the child has sufficient residual hearing to hear a human voice by properly amplified device, he may acquire speech and language."

7. "Children are classified as follows: profoundly deaf; perception of musical tones; perception of words and connected language. There is difficulty in placing children—general ability is an important factor."

8. "Only a slight amount of hearing makes the difference between a bewildered expression and one of quick eager interest."

9. "The child with 20 per cent of normal hearing has an advantage over the totally deaf one. The influence of even a little residual hearing cannot be overemphasized. The percentage varies with the ability of the child and the power of the hearing aid."

10. "Practically all children with residual hearing are more alert, more responsive and more normal because of it."

11. "We get better quality of voice and more natural speech if the child has some usable hearing."

Summary.—All seem to agree that children with 20 to 25 per cent residual hearing may make notable improvement in their speech patterns. The smallest amount of residual hearing may be of some possible use.

DISCUSSION

Dr. Fletcher D. Woodward, Charlottesville, Va.: I am grateful to Dr. Nash for having chosen the term residual hearing. I found it difficult to define. The development of this potential residuum is the portal through which the child is taught and his future life constructed. He mentioned the application of hearing aids to bring out this residuum. I think he should have mentioned other efforts, such as clearing up diseased conditions in the nose, throat and eustachian tube when this will indirectly help.

The paper must be read in detail to realize the amount of work done and the value of the conclusions. A survey will be helpful in classifying these children and in intelligently discussing the future plans for the individual child. There is also the importance of this hearing in the development of speech. A child who acquires normal rhythm and accentuation of speech has a great advantage over the child who does not.

Dr. Walter Hughson, Abington, Pa. (with slides): At the end of paragraph 2 slide 1 was shown. When the hearing reaches the level of 2048 double vibrations, the hearing loss is not as great as when the hearing is limited to 1024 double vibrations or lower. The child auricularly trained is superior in all factors concerned in articulation.

At the end of paragraph 3 slide 2 was shown. I presume that situation exists universally. It is the case in Pennsylvania and, I suppose, in other states. It is deplorable.

DR. EDMUND P. FOWLER, New York: This is an excellent paper, but it demonstrates one of the careless methods of reasoning—that of accepting the threshold audiogram as though it meant that that is the way the child hears. It is not the way the child hears. It is the way the child hears the threshold sound, and that is all it is.

The majority of Dr. Nash's cases were probably instances of nerve deafness, but in many of them obstructive deafness was combined with nerve deafness. You cannot test the ability to hear words by threshold sounds. The sounds must be amplified to where the words are intelligible to the ordinary human ear and even further for the hard of hearing child, because this child has to substitute for the sounds he does not hear the sounds he does hear. He usually hears better the sounds in the lower part of the scale. The patterns of the lower part of the scale are used to guess the ones in the upper part of the scale. That is the way he learns to hear and that is the way he is taught to hear.

The percentage on the slides certainly does not mean how much of speech that child heard. It means only so many decibels of hearing by threshold measurements.

DR. C. STEWART NASH, Rochester, N. Y.: I agree with Dr. Woodward that infection should be cleaned up and such benefit derived thereby as is possible. Only a small percentage of these children have otitis.

I also agree as to the importance of speech development. I have great respect for Dr. Hughson's work with the auricular method. He uses the word auricular and the word aural. I think the word visual would be better, because lip reading and finger spelling to me are visual.

I agree with Dr. Fowler that the percentage loss for the threshold sounds means nothing and that hearing words is a complicated affair, but I challenge him to give me a better method.

I again quote Dr. O'Connor: "Much better help is rendered children by regarding them as acoustically handicapped rather than deciding at the beginning that they are either deaf or hard of hearing and prescribing alone on the basis of the one or the other."

Review of Methods Used for Estimating Percentage Loss of Hearing.

MR. HOWARD A. CARTER, Chicago.

I have been led to the conclusion that a practical procedure for estimating percentage loss of useful hearing should fulfil certain requirements. It should be (1) easily understood, (2) easily and quickly applied, (3) free of complicated mathematical calculations, (4) easily interpreted before any jury, (5) designed for both ears jointly considered as a single hearing component, (6) based on air conduction data obtained from standardized audiometers, (7) weighted to give preference to the frequency range of the spoken voice and (8) founded on the best available acoustical and clinical scientific evidence.

In legal proceedings for damages to the hearing, the method deals only with one small but important part, i. e., the determination of the functional percentage loss of hearing. The method should be uncomplicated and designed for application to all persons in all walks of life. The fact that a hearing aid would improve the ability to understand speech does not enter into this text. The compensation allowed is determined by the court. For example, a piano tuner and a carpenter would have different degrees of handicap by deafness.

Sound travels through the air at a velocity of approximately 1,000 feet (305 meters) per second and in straight lines if it is not interfered with by some obstruction. Its intensity diminishes inversely as the square of the distance. In an enclosure sound is reflected and absorbed. The characteristics of sound within

a room are unpredictable, and the intensity of examiners' voices is a varying factor. Data on bone conduction are of no value in this method, however valuable they may be for diagnosis.

Eleven methods will be described for criticism herein. There is no scientific evidence by which to fix the combined loss of hearing of two ears into one percentage figure. For example, in regard to a person totally deaf in one ear with normal hearing in the other, a survey conducted by the Council revealed estimations ranging from 5 to 50 per cent. When an audiometer is used for determining hearing loss, explaining the meaning of a decibel to a jury may be regarded as a major pedagogic effort for a technical witness.

Army Method, 1940.—The acuity of hearing is determined by testing with a low conversational voice and by testing with the audiometer when indicated. The registrant is 20 feet (6 meters) distant, with the ear to be tested toward the assistant and the opposite ear closed by pressing the tragus closely to the meatus; the registrant is directed to repeat promptly the words spoken. If he does not hear at 20 feet, the speaker approaches foot by foot, using the same tone of voice, until the words are repeated correctly. A grade of 20/20 indicates normal hearing. If the registrant hears at 10 feet (3 meters) what the person with normal ears hears at 20 feet, his hearing is graded as 10/20. Comment: This is a simple workable method, but it is not designed for estimating percentage loss.

Navy Method, 1938.—The whisper test begins at 15 feet (4.5 meters). If the applicant does not hear, the assistant approaches foot by foot. Each ear is tested separately. A grade of 15/15 indicates normal hearing, and fractions of 15 are used to indicate hearing loss. The watch with constant ticking strength may be used with a denominator of 40 inches (100 cm.), which is the standard distance. Comment: This method does not fulfil the proposed requirements.

Veterans' Administration Method.—The acuity of hearing is determined by the voice test and the result checked by the audiometer when the latter is available. The patient is seated. Distances of 20, 15, 10, 5, 2 and 1 foot (6, 4.5, 3, 1.5, 0.6 and 0.3 meters) are used, the examiner whispering first and then using a conversational voice. Bone conduction is also determined by tuning forks. Comment: This method is not designed for determining percentage loss.

Conversational Voice Method Proposed by Dr. Henry H. Kessler.—Normal hearing is regarded as 20/40—i. e. hearing a conversational voice at 20 feet. Total loss of bone conduction is considered total deafness. Most states allow 40 per cent total permanent disability to be considered as complete deafness. Hearing at 20 feet with both ears is considered to indicate no loss of hearing. Hearing a conversational voice at 1 foot by each ear is estimated as a 20 per cent loss. Comment: The conversational voice is not constant, and therefore the method is not accurate in control. It is not explained how the table was compiled. The method is simple but does not fulfil the stipulations.

The following methods employ the audiometer. It is assumed that a judge or a jury will have some notion regarding the meaning of a decibel and will know that the audiometer measures hearing loss in sound units.

Fletcher Method.—The average decibel loss for 512, 1024 and 2048 cycles per second is multiplied by a factor, 0.8, to obtain the percentage figure. The method is extremely simple to understand, assuming that the meaning of the decibel and the makeup of the audiometer are understood. It deals with but one ear at a time and does not consider a joint percentage for the two ears. Comment: It does not fulfil the aforementioned stipulated requirements.

Maico Method.—The charts represent 20, 40, 60, 80 and 100 per cent curves. Each point on the curve is based on the data presented by Dr. Fletcher. Comment: The same criticism applies to this method.

Sonotone Method.—The Sonotone chart contains a series of curves marked 80, 60, 40, 20 and 0 per cent, but the percentage figures mean percentage hearing instead of percentage loss. Points on the curve are determined by calculating

the average decibel loss at 512, 1024 and 2048 cycles by multiplying by 0.8 and subtracting this from 100 per cent. Comment: It does not meet the stipulated requirements.

Kenny Method.—This is a combination of whisper and audiometric tests. The decibel losses at the frequencies 128, 256, 512, 1024, 2048 and 4096 as determined by the audiometer are called pure tone audiometer readings. The reading for the whisper test is obtained by whispering through a microphone connected to the audiometer through a calibrated rheostat, and the reading becomes part of the data. The average decibel loss for the six frequencies is multiplied by the factor 1.2. The average audiometric reading is added to the loss for the whispered voice, and again the numbers are averaged. The result is the percentage loss for one ear. It is estimated that a 100 decibel loss for the whispered voice is total loss of hearing. Comment: The whisper test varies with different subjects. There is no standard method for calculating a whisper through an amplifier and a rheostat. It is doubtful whether this procedure could be explained to a jury. Since there is no joint percentage figure for the two ears, the method does not meet the requirements.

Fowler Method (Fowler, E. P., Jr.: Medicine of the Ear, New York, Thos. Nelson & Sons, 1939, p. 419).—This method is based on air and bone conduction losses combined at the frequencies 256, 512, 1024, 2048 and 4096. The average loss is obtained by dividing the total by 10. The percentage loss in the better ear for each frequency is indicated along horizontal lines; the percentage loss in the worse ear is indicated along vertical lines. The total percentage is located where the two lines meet. This method is simple for the operator but it would be difficult to explain before a jury how the percentage figures are determined. The method is also open to criticism because data on bone conduction are used. Bone conduction has not been standardized.

Fowler Method (Fowler, E. P. Jr.: A Method for Measuring the Percentage of Capacity for Hearing Speech, J. Acoust. Soc. Am. **13**:373 [April] 1942).—The figures indicating decibels of threshold loss are weighted as follows: at 512—15 per cent; at 1024—30 per cent; at 2048—40 per cent; at 4096—15 per cent. The products are added and divided by 100. This is the weighted decibel loss when there is no nerve deafness. If the loss by bone conduction is 15 decibels, a recruitment factor, 6, is subtracted from the threshold loss by air conduction before multiplying by the weighted figure. For each 5 decibels of loss more than 15, deduct 1 decibel less for the recruitment factor before multiplying by the weighted figure. The percentage is arrived at as in the preceding method. Comment: One less frequency is used and four frequencies are weighted according to their value in hearing speech. The criticism stated in the foregoing paragraph applies to the use of bone conduction in this method. The evidence to substantiate the selection of percentage numbers is not clearly defined.

Sabine Method.—The audiometer chart is ruled off into small rectangles, having boundaries on the horizontal lines at 128, 256, 512, 1024, 2048 and 4096 cycles per second. The vertical boundaries represent 10 decibel steps. Each rectangle has a number which represents the weighted percentage loss for a corresponding region in the sound spectrum. For example: The useful hearing for the right ear is determined by adding the numbers in the rectangle immediately above the audiogram curve, and the sum represents the percentage weighted loss in the region between the two octave frequencies. The same is done for the five columns; i. e., between 256 and 512; 512 and 1024, and so on. The same process is repeated for the left ear. To obtain a combined percentage loss of hearing, the total percentage loss of the worse ear is added to one eighth of the loss of the better ear. Comment: The weighting may be questioned, but so far no one has suggested a better factor. Any method submitted to the otologic profession would be subject to considerable criticism and rightfully so. The method, if sound, should stand up under critical examination.

Requirements for a practical method to estimate the percentage loss of hearing have been presented, and eleven methods have been reviewed. The speculative outline is limited to medicolegal or industrial cases only and is not intended for diagnostic purposes.

DISCUSSION

DR. EDMUND P. FOWLER, New York: Mr. Carter has clearly presented several methods for estimating the percentage hearing but has passed over many of the difficulties.

The phenomenon observed in certain types of nerve deafness—the loss of hearing for articulation without a corresponding loss for the frequencies used in hearing speech, or a loss of memory of the sound of words—deserves considerable thought and discussion. Two ears may have identical threshold audiograms for air and bone, yet one ear is able to understand the spoken word when this is sufficiently amplified and the other cannot understand it no matter how much the voice is amplified. Hearing aids are of no avail. I have never observed it in the most severe obstructive deafness unless this was complicated by nerve deafness of over 45 decibels for the speech frequencies. It may occur in nerve deafness when hearing for speech frequencies is down only 45 to 50 decibels. In these cases the recruitment phenomenon is present and bone conduction markedly impaired.

The phenomenon has been observed in people with ipsilateral labyrinthine disturbances and frequently in persons with tinnitus, which is constant and difficult to mask. It is possible that both the tinnitus and the deafness are due to central lesions or to some now unknown pattern of degeneration of the cochlea. It is improbable that the lesion is cortical, as only one cortex is necessary for hearing speech, or that it is a psychoacoustic asthenia affecting one ear only.

The deafness for speech differs from those types of deafness that are due to marked losses limited to the upper part of the scale, as in them it is possible to make speech more intelligible by a proper combination of hearing aid frequency-loudness controls. The brain seems to be able to interpolate the missing high tones or to conjure them up from the low tone patterns. High frequencies do not need to be heard as clearly as low to be useful in hearing speech. It may be that the brain shuts off the hearing in the poorer ear when distortion of speech interferes with the hearing in the better ear. The following experiment is illustrative: Listen to two radios receiving the same broadcast, one with good radio reception opposite the right ear, the other with poor, scrambled reception at the left ear. It is natural to concentrate on the ear hearing the good reception and to exclude the ear hearing the poor reception. The louder the jumbled sound, the more it masks acoustically and psychologically the sounds one desires to hear. These phenomena must be kept in mind in estimating the percentage capacity to hear speech.

A careful study of serial sections of the auditory tracts and nuclei in subjects who have been properly examined clinically will be of service in solving this mystery of speech deafness without cortical lesions. I have asked the help of Dr. S. J. Crowe and Dr. Stacy Guild, of Columbia University, in the solution of this problem.

DR. RALPH FENTON, Portland, Ore.: Permit me to say on behalf of the Board of Trustees of the American Medical Association that the Board has been deeply sensible of the value of the ophthalmologic standards adopted after careful study by the Council on Physical Therapy.

For the benefit of courts, of the United States Veterans' Bureau and the Bureau of Legal Medicine of the American Medical Association, we are anxious that some system be devised for the accurate determination of percentage loss of hearing which may be admissible under the rules of evidence in courts. It is important that it be received by the various industrial accident commissions, and the matter has been discussed in the Council on Industrial Medicine.

If the Council on Physical Therapy gives us some system and it meets with the approval of this society, it may then receive the necessary approval of the Section on Laryngology, Otology and Rhinology of the American Medical Association. Then, I am sure, it will receive the unanimous approval of the various industrial accident commissions, as did the ophthalmologic standards which were placed in effect for a number of years.

DR. BENJAMIN H. SHUSTER, Philadelphia: I have the president's permission to present some figures from Dr. Robert Hunter, who is in the Service and unable to be here.

He proposes a method of calculating the percentage hearing loss at various distances, 20 feet being assumed to be normal, and the voice or whisper test being used. His calculations are based on the fact that the intensity of sound varies inversely as the square of the distance. Assuming that 20 feet is the normal, he squares the numbers, multiplies by 100 and extracts the formula in percentage. The final formula amounts to this: 100 minus $\frac{D^2}{4}$ (D meaning distance). Using a 10 foot hearing distance, the formula would be applied as follows: The square of 10 is 100; this divided by 4 equals 25, and this subtracted from 100 equals 75 per cent loss. This becomes more controversial when one gets down to 5 feet: 5 squared is 25; this divided by 4 equals about 6; this subtracted from 100 gives 94. In other words, when the subject hears at 5 feet what he should have heard at 20 feet, there is a loss of 94 per cent.

This does not seem practical to me, but he insists that it is. He seems to think I am wrong. He asked me to present these figures, and I have done so, although I do not agree.

He has also calculated the decibels of loss in terms of logarithm. He gives the log of decibels with the foregoing formula. He boils that down to the result that every time the distance is halved or doubled, there is a change of 6 decibels.

DR. WALTER HUGHSON, Abington, Pa.: In regard to Mr. Carter's method, no mention is made of testing the ability to understand speech. This is the test all otologists are interested in. During the past few years my associates and I have been using a method to measure improvement following therapy and to fit hearing aids.

It has been possible to establish a normal threshold for speech reception by examining a sufficient number of normal young persons by means of measured controlled intensities of speech, and thereby the relationship of a deaf person's hearing to normal hearing and understanding of speech. To our surprise this percentage audiometric loss bears a direct and constant relationship to speech reception. That relationship is in the order of 2:1. I wonder if it is not actual measurement of one's ability to understand speech which is the practical thing and not the effort to try and fit the audiogram into it.

DR. ALFRED LEWY, Chicago: Item 5 of Mr. Carter's first slide indicated that tests were to be made of the hearing of both ears together. The state of Illinois and I don't know how many others pay compensation for total loss of hearing in one ear. What is meant by total loss has not yet been passed on by the highest court, but arbitrators are inclined to accept as total loss, loss for industrial purposes.

There is another complexity that applies to all of the present tests. Lawyers try to keep out an expert's opinion on hearing from the standpoint that the tests are subjective. If the Court is sensible, this is overcome by his asking whether the tests are those usually adopted by otologists for determining the degree of hearing. Acceptance does not always go through on that premise.

I agree with Dr. Fenton that courts will welcome any method that is approved by an authentic body like the Council of Physical Therapy of the American Medical Association or the United States Bureau of Standards and by physicians. They will, I believe, adopt it over the quibbling of the lawyers whether it is subjective or not.

MR. HOWARD A. CARTER, Chicago: The Consultants on Audiometers and Hearing Aids are really the gentlemen who have been working hard to get a solution, and those who are present no doubt appreciate hearing these comments.

Dr. Fenton pointed out, and I wish to urge, that otologists should give careful consideration to any method which may be submitted, because after all it must stand on its own feet.

I did not get Dr. Shuster's method down fast enough and am not able to comment on it. I did not hear him say that both ears were taken collectively. Hence I do not think the method would fit into the general scheme.

As an invited guest I could not go into all of the problems which were considered, so I left out the understanding of speech referred to by Dr. Hughson. I can assure you that considerable thought has been given to that problem.

Dr. Lewy asked whether there is a definition of total loss of hearing. Yes—it has even been placed on an audiogram. Essentially it means that if a person cannot understand the spoken voice in his native language shouted within 6 inches or 1 foot from the head, that person is practically totally deaf as far as the industrial point of view is concerned.

(To Be Concluded)

Book Reviews

War Medicine: A Symposium. Editors: Winfield Scott Pugh, M.D.; Edward Podolsky, M.D., and Dagobert Runes, Ph.D. Price $7.50. Pp. 565. New York: Philosophical Library, Inc., 1942.

This book represents an honest attempt to gather under one cover the "cream" of a vast volume of literature dealing with war medicine in all its phases which has appeared during the past three or four years. The editors have succeeded fairly well, although much, of course, had to be omitted. A whole series, encyclopedic in size, would be required to cover the subject in its entirety. Moreover, every day adds to knowledge of this all-engrossing and important matter.

The table of contents comprises three main divisions: surgery, aviation and naval medicine, and general medicine. To begin with, every physician should, indeed must, read the chapter on "War Wounds," by Dr. John J. Moorhead, page 24. In approximately 10,000 words Dr. Moorhead has put into a delectable capsule about all the theoretic (and practical) knowledge which might be helpful in a "refresher" course. It is a working outline covering the salient points in emergency surgery and is, in truth, an amazing performance, embracing as it does the heart-rending experiences of Pearl Harbor.

There follows some 13 pages dealing with anaerobic infection of war wounds, by Major R. I. Pritkin, of the Medical Corps, Thirty-Third National Guard Division, who has made a special study of gas gangrene. He says that anaerobes succumb quickly to hypochlorite solutions and to chloramine-T. One would expect extended comment on the use of oxygen, both in gaseous form and as hydrogen peroxide, but it is mentioned in passing only. The same is true of roentgen rays, light and heat. However, laboratory studies of many types of anaerobes are described, including the effects of free oxygen on Bacillus Welchii, clostridium tetani, Bacillus botulinus, Bacillus putrificus, Bacillus oedematiens and Bacillus histolyticus.

Dr. Walter Wells, of Washington, D. C., describes the danger to hearing in modern warfare, particularly in the Air Corps. He paints a true, but necessarily gloomy, picture of loss of hearing which is sure to be the lot of postwar veterans. High altitude flying and dive bombing at 400 miles (640 kilometers) per hour are likely to damage or destroy the organ of Corti, and those who escape will be lucky indeed.

Dr. Alfred B. Alexander discusses rupture of the "drum head" in warfare, and very properly advises no meddling. He blows in sulfanilamide powder, but finds that it runs away in the discharge! One wonders whether he has tried the boric acid–iodine powder of Sulzberger. It is a specific remedy of sovereign value in cases of this kind. Alexander makes the point that all air raid victims should be examined otoscopically.

Chapters in this book deal with pilot fatigue, with blood bank and transfusion methods and with use of anesthesia at the "front," all of them of interest and importance to every physician who works in or out of war zones.

The ophthalmo-otorhinolaryngologist whose experience has been confined to civilian practice will be greatly surprised to find how little he knows of the basic facts in aviation medicine. If he thinks air surgeons are "fussy" about such matters as nasal obstruction, acute or chronic sinusitis, color blindness, depth perception, a discharging ear or "slightly impaired hearing," he will not go far in his reading before the reasons appear as stark and naked truths. For example, a flyer who is taking any of the sulfonamide drugs must be grounded for the time being. Reason: anoxia. If he is color blind, that broad expanse of green grass may turn out to be a lake. If his colon is loaded with gas, he may blow up at

20,000 feet (6 kilometers), literally. If he has a cold or other nasal infection or obstruction, he may suffer from hemorrhages into the mucous membranes and tympanic cavity. All these things, and many more, can be learned from a careful reading of this and other books of the kind. Those specializing in otolaryngology are being drawn deeper and deeper into the field of "general medicine" during these awful days. It behooves each one to read all such books as "War Medicine."

Textbook of the Ear, Nose and Throat. By Francis L. Lederer, B.Sc., M.D., F.A.C.S.; Professor and Head of Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine, and Abraham R. Hollender, M.D., F.A.C.S., Assistant Professor of Laryngology, Rhinology and Otology, University of Illinois College of Medicine. Third edition. Price $10. Pp. 894, with 730 illustrations, 18 colored. Philadelphia: F. A. Davis Company, 1942.

The authors present a textbook for medical students that is in keeping with modern trends. Revision of teaching rosters and schedules has become necessary because of the accelerated medical course.

This textbook, as outlined in the preface, aims to supplement the lectures and quizzes, and in the 595 pages it is obvious that the authors have succeeded in their aim and purpose. There are seventy-two chapters, covering in detail all phases of otolaryngology.

The authors have introduced a novel idea in this textbook, and it may well serve as a basis for similar ideas in other textbooks. This idea is briefly described thus: At the head of each chapter is a "preview," which consists of a condensed, comprehensive description of the subject of that chapter in about ten to fourteen lines, usually forming two paragraphs. It is a brief, yet full, explanation of the discussion about to be presented and serves as a perspective of the chapter about to be discussed. The entire preview is enclosed in a square.

This reviewer will quote verbatim from the preview of chapter 1, entitled "Changing Trends in Otolaryngology."

"During the past half century enormous progress has been recorded in the development of the ear, nose and throat specialty. Increasing knowledge of otolaryngologic diseases and improved apparatus and instruments have contributed to the rapid advances in diagnostic methods. The modern specialist must have a comprehensive knowledge of pathology. Adequate ventilation and drainage remain the important principles of non-surgical or surgical treatment in rhinology. More determined efforts are now being made to practice otolaryngologic surgery on a physiologic basis. Therapeutic advances in surgical conditions can be laid to this factor, refinement of operative technics and chemotherapy."

All the "previews" are as clearcut and comprehensive as the one just outlined.

The textbook is complete in every detail. The descriptions and discussions of the various subjects in a language that is as close to grammatical purity as one is likely to find, augmented by first class illustrations, drawings, photographs, photomicrographs, line drawings and schemas, make this text an outstanding contribution.

The format, binding, printing and arrangement of the chapters merit glowing praise.

The authors should be congratulated on this contribution to American otolaryngology, a textbook that has been presented during a time of stress, hurry and much activity.

There is little doubt that many medical students will use this book liberally and find much to supplement their lecture outlines.

It is strongly recommended to medical students and to all those in the practice of medicine and surgery who are likely to encounter otolaryngologic problems during the routine, if not intensive, studies of their cases. It should also be a valuable reference book for the clinic and for hospital wards.

Manual of Standard Practice of Plastic and Maxillofacial Surgery. Prepared and edited by Subcommittee on Plastic and Maxillofacial Surgery of the Committee on Surgery of the Division of Medical Sciences of the National Research Council, and Representatives of the Medical Department of the United States Army. Robert H. Ivy, Chairman. Price $5. Pp. 432, with 899 illustrations on 259 figures. Philadelphia: W. B. Saunders Company, 1942.

This is the first in the series of the war manuals to be published by the Division of Medical Sciences of the National Research Council. The volumes on the eye and ear and abdominal and genitourinary surgery are also ready. There are two more on other subjects yet to come. This volume is divided into four sections. Section I consists of ten chapters, which are concerned with general considerations, condensed discussions, cheiloplasty, meloplasty, rhinoplasty, blepharoplasty, otoplasty, defects of the scalp and cranium, cervicoplasty and loss of the hard palate and the premaxillary portion of the alveolar process. The excellent drawings and sketches in this section elucidate the text completely. The latter is well written, simple, direct, forceful and wholly understandable. These chapters contain the essence of all that the surgeon must know in dealing with severe injuries and loss of tissue.

Section II is brief, containing two chapters concerned with the jaws in relation to gunshot wounds and fractures. This is a field in which the dentist has hitherto labored ardently and ably. The making of molds, casts and splints, wiring of the mandible, etc., are somewhat outside the area of most general or special surgeons, but by following the outline given here one may work out the technic when no dentist is at hand.

Section III defines and describes maxillofacial prosthesis, which is decidedly a task for the artist accustomed to deal with molding compounds. This is the kind of work that requires much time and patience and is for the most part not "emergency."

Section IV is concerned with anesthesia technics. Local anesthesia is of three varieties: infiltrative, topical and regional. In the part on general anesthesia, preoperative medication is described. Then the matter of choice of agent is taken up, and, finally, the postoperative care of the patient is considered. A satisfactory index completes the volume.

Having read and reviewed the first three "Manuals," this annotator regards this first volume as the best so far produced.

Surgery of the Nose and Throat. By John D. Kernan, M.D., Editor; Professor of Otolaryngology, Columbia University College of Physicians and Surgeons; Attending Otolaryngologist and Bronchoscopist, Lenox Hill Hospital. Price $15. Pp. 701, including index, with 616 illustrations and many charts and tables. New York: Thomas Nelson & Sons, 1942.

"Surgery of the Nose and Throat" is the title of the latest one volume text in loose leaf form presented by Thomas Nelson & Sons. Dr. John D. Kernan is the editor.

The aim of the editor and the contributors has been to prepare in one volume a systematic and complete text that would contain all the necessary data on the surgery of the nose and throat. The surgical procedures advocated are, as far as possible, those calculated to preserve structure and restore function. Each contributor has been painstaking in his assignment and has fully carried out the planned program.

There are in all 701 pages, each in double column; the text material is well arranged and profusely illustrated with charts, drawings, schemas and photographs, so that the reader can carry out his search for any surgical procedure or discussion to the finest detail. Abundant references and a good index are of great help.

The value of this text can also be judged by the eminence of the contributors, each one being competent and thoroughly skilled in his field: J. D. Kernan, Harry

Neivert, Henry B. Orton, C. L. Jackson, A. A. Eggston, J. B. Brown, G. R. Brighton, Hayes Martin and Arthur J. Cracovaner.

This volume is recommended to otolaryngologists, oral surgeons, residents and graduate students in otolaryngology.

Nasal Medication: A Practical Guide. By Noah D. Fabricant, M.D., M.S.; Associate in Laryngology, Rhinology and Otology, University of Illinois College of Medicine. Price $2.50. Pp. 122, with 20 illustrations. Baltimore: Williams and Wilkins Company, 1942.

The author had a twofold purpose in mind when he wrote this book: to make it a practical guide and a reference work. It is a small volume, but 122 pages in all; yet it covers a large field. In ten chapters, dealing with the anatomic, histologic and histopathologic features and physiology of the nose, the effect of drugs on ciliary action, nasal p_H and nasal mucosa, methods of applying nasal medication, nasal medication in treatment of disease and nasal medication in children, with many illustrations and photomicrographs, a vast amount of valuable data is presented.

The entire text is well written, the advice sane and authoritative and the bibliography sufficient to enable the reader to delve further into the subject under discussion. The author and subject indexes are complete.

This book is recommended to all physicians employing nasal medication in their various fields.

News and Comment

CASSELBERRY AWARD OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION

A sum of money is now available from the Casselberry Fund of the American Laryngological Association for a prize award, decoration or grant to be given for original investigation in the art and science of laryngology or rhinology. Theses must be in the hands of the secretary, Dr. Arthur W. Proetz, 1010 Beaumont Medical Building, St. Louis, before March 1, 1943.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Claude C. Cody, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. Robert L. Moorhead, 125 Ramsen St., Brooklyn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James G. Dwyer, 375 Park Ave., New York.
President-Elect: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. Edward J. Whalen, 750 Main St., Hartford, Conn.
Place: Hartford, Conn. Time: Jan. 15, 1943.

Southern.—Chairman: Dr. Francis B. Blackmar, Swift Kyle Bldg., Columbus, Ga.
Place: Chattanooga, Tenn. Time: Jan. 28, 1943.

Middle.—Chairman: Dr. Voss Harrell, Hofman Bldg., Detroit.
Place: Detroit, Mich. Time: Jan. 20, 1943.

Western.—Chairman: Dr. Irving M. Lupton, Medical Arts Bldg., Portland, Ore.
Place: Portland, Ore. Time: Jan. 31, 1943.

American Otological Society

President: Dr. Wesley C. Bowers, 17 E. 61st St., New York.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of sccieties are requested to furnish the information necessary to keep this list up to date.